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THE TREATMENT OF BRIGHT'S DISEASE AND RELATED RENAL INFECTIONS*

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IN the management of the various forms of Bright's disease, with rare exceptions, we can neither attack the underlying etiologic causes nor treat the pathologic processes in any fundamental way. We deal mainly with individual symptoms and we attempt to maintain the proper physiologic balance of the body. In so doing, much can be done not only to enhance the comfort of the patient but also, in many instances, to ameliorate the course of the disease.

In discussing the management of the various forms of Bright's disease, I shall divide these manifestations broadly into those that tend to be acute and those that are chronic in nature.

ACUTE NEPHRITIS AND PYELONEPHRITIS

Acute Nephritis. The general principle of handling patients with acute hemorrhagic or inflammatory glomerulonephritis, seen chiefly in children and young adults, is rest for the individual and, so far as is possible, for the kidneys. Such patients should be put to bed and should remain there until the recovery processes have been accomplished or the disease has definitely become chronic. Recovery can be considered to have been attained when symptoms have disappeared, when the blood pressure has returned to a normal level, when there is a normal sedimentation rate and when the albuminuria and hematuria have disappeared or have decreased to a minimal level. During the first few days, in the severe cases, fluids should be markedly restricted, and the diet limited mainly to carbohydrates in liquid form. Later, the diet should be one that is easily assimilated and that contains an adequate number of calories. Recent work by Keutmann and McCann[1] has indicated that there is no evidence that a high protein diet increases the kidney irritation in such cases. In practice, it is not necessary to give large amounts of protein in acute nephritis, nor is it necessary or desirable to restrict the protein intake markedly, except in cases of impending uremia. Fluid intake should be ample but not excessive, about 2 liters daily being a proper average amount; salt may be restricted during the early stage, especially if there is edema.

It has been shown that most cases of acute glomerulonephritis are related to streptococcal infections. Foci of such infections, particularly those in the tonsils and sinuses, should, therefore, be assiduously sought and, if found, removed. It is probably wisest to remove frankly infected tonsils or other undoubted foci of active infection as soon as possible in the course of the disease, although some authorities counsel against tonsillectomy until the initial stage of the nephritis has passed, because of the danger of increasing its severity by such an operative procedure. It has been noted that those cases of acute nephritis that follow an undoubted streptococcal infection are most likely to run a benign course and to clear completely, whereas those that begin insidiously in a patient in whom a focus of infection is not readily apparent usually do badly and progress to the subacute and chronic stages.

To prevent recurrences of nephritis, patients should be urged to make every effort to guard against acute infections, particularly those of the respiratory tract, by attention to general hygiene and by avoiding exposure, and to treat such infections vigorously, if they do develop.

It has been shown by Blackfan and his coworker[2] that the alarming cerebral manifestations (intense headache, vomiting, convulsions, coma) which occasionally ensue in the severe cases of acute nephritis are usually due to cerebral edema rather than to uremia. Indeed, nitrogen retention is often absent at such times. The best index of impending cerebral edema is to be found in a progressive, rapid rise in the blood pressure. The proper treatment, which is usually very effective, is to give magnesium sulfate, by mouth, in doses of 30 to 60 cc. of a 50 per cent solution every 3 or 4 hours. If this is ineffective, then the magnesium sulfate should be given intramuscularly in a dose of 0.2 cc. of a 25 per cent solution per kilogram of body weight. Such a dose can be repeated in 3 hours if necessary. Recent work[26] has indicated that intravenously administered sucrose may be very effective in combating increased cerebrospinal fluid pressure, and since it is probably a safer preparation than magnesium sulfate, it may

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prove a valuable therapeutic measure in this condition.

Urinary Infections. A very considerable number of patients who are supposed to be suffering from chronic glomerulonephritis or malignant hypertension have in fact a nephritis secondary to a bilateral pyelonephritis of long standing.[3, 4] The clinical recognition of such cases may be difficult, for often the patients have never had acute attacks severe enough to bring them under observation. They usually consult a physician only at a time when renal failure, namely, uremia, is already setting in. Longcope and Winkenwerder[4] have emphasized that a careful consideration of the past histories of such patients frequently reveals that they have had repeated febrile episodes and also that not uncommonly they have complained of chronic back pain. Renal edema in these individuals is rare. Examination of the urine shows a low fixed specific gravity, relatively little albumin and few casts, but considerable numbers of leukocytes.

There are two aspects in the treatment of such individuals. The first is the recognition of the facts that renal failure may ensue as a terminal stage in pyelonephritis and that it is necessary, therefore, to treat vigorously the pyelonephritis in its early stages; and the second is the treatment of the condition, when it is once established, in the late stages.

It has been shown that most of the cases of acute pyelitis in infants and children,[5] and probably in adults as well,[6] who do not have ureteral obstruction, have abscesses and foci of inflammation in the kidney parenchyma. Since the difference between the clinical cases of pyelitis and of pyelonephritis is one of extent of involvement and of degree of chronicity, it is advisable to classify all such cases as acute or chronic pyelonephritis.

In the treatment of acute pyelonephritis or pyelitis, rest in bed and the forcing of fluids should first be instituted. If the condition does not clear up promptly, or if there is a tendency to relapse, then further measures are in order. It has come to be the general opinion that most urinary antiseptics by mouth are of little or no value. The most promising treatment for persistent colon bacillus infections of the urinary tract is the use of either the ketogenic diet or mandelic acid. The use of a ketogenic diet for this purpose was first emphasized by Clark[7] and by Helmholz[8] at the Mayo Clinic. A number of reports from various sources in this country and abroad have all attested to its success; about 70 to 80 per cent of uncomplicated cases of bacilluria became free of the infection. The effectiveness of the treatment rests apparently in two factors: the production of a markedly acid urine and, as shown by Fuller,[9] the specific bacteriostatic action of the levorotatory beta-oxybutyric acid that is excreted. Successful treatment, therefore, consists of acidifying the urine as much as possible and in creating an adequate ketosis. In vitro it has been shown that the growth of colon bacilli is inhibited at a pH of from 4.6 to 5.0.[7] Actually, in the presence of the beta-oxybutyric acid the urine need not be acidified to this degree, but it should be kept below a pH of 5.5,[8] a point that can be easily measured clinically by the use of chlorphenol red as an indicator. If the ketogenic diet alone does not produce this degree of acidity, the addition of ammonium chloride to the diet in amounts of from 6 to 10 grams daily should be carried out. Recently the use of nitrohydrochloric acid as an acidifying agent has been advocated.[10] This should be given in doses of 10 minims four times daily. The ketogenic regime must be maintained for from 1 to 3 weeks. The details of the practical administration of the ketogenic diet have been well discussed by Talbot.[11]

Recently, mandelic acid has come into use in the treatment of persistent urinary infections, especially those due to the colon bacillus. Rosenheim[12] first advocated the employment of this substance, which is chemically related to beta-oxybutyric acid and which apparently has a specific bactericidal effect, as well as being a powerful acidifying agent. Cook and Buchtel[13] of the Mayo Clinic found that its use proved effective in rendering the urine sterile in from 50 to 81 per cent of cases of bacillary infection. They administered 12 grams of sodium mandelate daily in divided doses, together with the usual acidifying drugs, such as ammonium chloride, and restricted the fluid intake to 1,200 cc. If the ammonium salt of mandelic acid is given, no other acid drug is usually required. These authors, who worked with patients, as well as Helmholz and Osterberg,[14] who carried out experimental work on dogs, have found that a urinary concentration of mandelic acid of 0.5 per cent or more is required, and the pH of the urine must be kept below 5.5. Other writers[15, 16, 17, 18] have confirmed the beneficial results of the drug and have found that its use is usually effective against colon bacillus infections in from 4 to 12 days, but that it is of much less value in infections with other organisms. Caution as to the untoward effects of mandelic acid is necessary. Diarrhea occurs in 10 per cent of the cases, and nausea is encountered in about 1 per cent.[13] More important, however, is the fact that the drug may produce irritant or toxic effects on the kidneys, as evidenced by temporary depression of renal function[13] or by the occurrence of casts or hematuria.[12, 18] Its use, therefore, in patients with markedly impaired kidney function is inadvisable. In general it may be concluded that mandelic acid is a valuable adjunct to the therapeutic armamen-

tarium against renal infections; that, because of its comparative ease of administration, its use is preferable to that of the ketogenic diet in the majority of instances (although application of the two methods jointly is sometimes advisable); but that the indiscriminate employment of the drug is to be deplored. Cases should be first carefully studied and selected, the proper amounts of the drug and of fluids must be given, and the clinical condition of the patient and the urinary findings must be repeatedly checked.

All cases of urinary infection that do not respond readily to medical treatment should be investigated carefully by pyelography to rule out conditions that might interfere with free drainage. If such conditions are found, and if they are remediable, they should be treated.

In considering the treatment of urinary infections it is important to remember that they have a tendency to be both persistent and recurrent, so that careful follow-ups over long periods of time are highly important. Through proper treatment of the pyelonephritis in its early stages, we can hope to arrest the process in the kidney in many instances, and thus prevent the disastrous end results of nephritis and hypertension, which occur not infrequently.

In the late stages of pyelonephritis, the general treatment is the same as that for chronic nephritis, with particular emphasis on trying to build up the patient's general condition by hygienic measures. The local treatment consists of making sure that both kidneys have free drainage by correcting surgically, any obvious faults, and by ureteral catheterization and pelvic lavage.

CHRONIC NEPHRITIS

From the point of view of treatment, chronic Bright's disease can be classified most simply according to the type of symptoms it presents. Its etiology is often obscure or undeterminable, and attempts to correlate its clinical aspects with the pathologic processes are unsatisfactory. A certain number of these cases of chronic Bright's disease, probably not over 10 per cent, definitely result from a known attack or attacks of acute inflammatory glomerulonephritis (hemorrhagic Bright's disease). Another group, undoubtedly larger than has been believed, is the result of renal changes secondary to chronic pyelonephritis, as has been previously discussed. Still another group is composed of patients who have vascular disease of the nature of benign or malignant hypertension (nephrosclerosis, arteriosclerotic Bright's disease). A few may be due to amyloidosis or to other degenerative causes, and this group may include the so-called "lipoid nephrosis" (cryptic degenerative Bright's disease) seen chiefly in children. There remain, however, a large number of cases in which no etiologic or exciting cause can definitely be demonstrated. It is usually assumed that such cases represent chronic diseases that have followed unrecognized acute glomerulonephritis, or at least those that are etiologically related to this type of nephritis.

In general, from the viewpoint of treatment, however, chronic Bright's disease can be divided into three stages: first, that which is latent; secondly, that in which renal edema tends to develop; and, thirdly, that in which nitrogen retention leads eventually to uremia. These subdivisions do not imply three separate diseases, for any given patient with chronic Bright's disease may and often does present at different times two or all three of these stages.

Chronic Latent Nephritis. In the latent stage there are either no symptoms at all or only minor symptoms. The patient, however, frequently presents evidence in varying degrees of peripheral vascular disease, such as hypertension, retinitis, cardiac enlargement and thickening of the peripheral vessels. The urine usually shows albumin in amounts ranging from small to large, and the sediment may contain red and white blood cells and casts. The renal function may be anything from normal to markedly impaired.

In the treatment of nephritis in the latent stage, attention should be directed primarily to the patient as a whole. Most of these individuals never develop uremia; a far larger number eventually die of heart failure. The general management of such patients should be aimed at building up the general body resistance by hygienic measures and by avoiding conditions leading to strain and tension, which may aggravate the vascular disease. The patient should be put on a schedule of living that will ensure him plenty of rest without too many restrictions, for since this is a chronic disease and the value of too limited a regime of activity is unproved, the happiness of the patient must be considered. All possible measures should be taken to avoid infections of all sorts, particularly respiratory infections, even the minor ones such as the common cold, since they may aggravate the renal disturbance and are in general the most prominent precipitating factors in heart failure. When such infections do develop, they should be treated vigorously with bed rest and other appropriate measures. Obvious foci of infection, such as those in the teeth, tonsils and sinuses, should be eradicated, but overenthusiasm in this respect should be avoided.

The diet should be chosen with the idea rather of providing essential substances, than of restriction. Aldrich and Boyle[19] found that giving a high vitamin diet resulted in striking improvement in a group of children with chronic Bright's disease. Although the rationale for such a procedure may not be very clear, never-

theless it does seem advisable to provide plenty of vitamins in the diet. In general the protein in the diet should not be restricted. Indeed, if much albumin is being lost in the urine, this extra drain must be made up by increasing the protein intake. Although each case presents a special problem, an average protein requirement for an adult patient is about 80 to 100 grams. The total caloric intake should be adjusted to maintain a satisfactory body weight. Marked degrees of underweight or overweight are undesirable—underweight, because it frequently betokens poor physical condition and resistance, and overweight, because of the extra strain on the heart and circulation that it imposes. Salt need not be markedly restricted so long as no tendency to edema formation exists.

Most patients with chronic Bright's disease have generalized cardiovascular disease. As has been already stated, their lives are more commonly terminated by heart failure than by renal failure, and cerebral vascular accidents or crises are of frequent occurrence. These events should be watched for, guarded against so far as possible and treated properly when they do occur.

Chronic Nephritis with Edema. Certain patients with chronic Bright's disease develop renal edema, the so-called nephrotic syndrome. This varies from a mild, intermittent edema to massive anasarca, resisting all attempts at therapy. Renal edema should always be distinguished from the dependent edema of heart failure, which is due to increased hydrostatic pressure, for the therapy of these two conditions is quite different.

The edema of acute nephritis is usually due to a generalized toxic effect on the capillaries with increased permeability. It is rarely severe and disappears with the subsidence of the disease, requiring no specific therapy. In the subacute stage, however, edema may occur as a result of the factors discussed below.

Since the factors leading to the formation of chronic renal edema are the same whether the underlying pathogenesis of the disease is chronic glomerulonephritis or lipoid nephrosis (degenerative Bright's disease), it is not necessary here to consider the differentiation of these two conditions nor to discuss the evidence for and against lipoid nephrosis being a specific disease. In this type of edema the most striking finding is a lowering of the blood plasma proteins, particularly of the albumin fraction. This is usually due to the loss of albumin, and occasionally of globulin, from the blood into the urine, although it may be aggravated by an undue restriction of protein in the diet.

The exact sequence of events and the nature of the pathologic processes that lead to the development of marked proteinuria and lowering of the plasma protein level are by no means clearly understood. It has not been definitely established whether the cause is a pathologic increase in the permeability of the glomerular epithelium allowing the normal albumin of the blood to pass through into the urine or whether, as certain recent work would suggest,[20, 21, 22] there is a qualitative biologic or chemical difference between the plasma proteins of normal persons and those of patients with the nephrotic syndrome, which may concern either the synthesis of these proteins or their diffusibility. For the sake of this discussion on treatment, however, it is sufficient to consider a simple loss of protein in the urine as the main cause of the lowered plasma protein and to evolve a rationale of therapy from this.

Normally, the proteins of the blood plasma are chiefly responsible for the difference in osmotic pressure between the blood stream and the tissue spaces—a condition that prevents fluid from accumulating in excess in the tissues. When the plasma protein is low, as in nephritis with edema, the osmotic pressure is reduced and edema tends to result. The plasma proteins can be readily estimated chemically, and such a test is of the greatest importance clinically in distinguishing edema of this type from other forms, since a plasma protein of 5.5 per cent or higher —the normal is 6 to 8 per cent—will not ordinarily produce edema,[23] whereas edema usually results when the protein is below 5.5 per cent. A more accurate indication of the plasma osmotic pressure is obtained by determining the albumin-globulin ratio of the plasma, as well as the total protein level. Normally, the albumin comprises about two thirds of the total proteins, but in patients with renal edema, the ratio is reversed and the albumin is usually below 2.5 per cent.[23] A simple test for estimating whether the proteins are above or below the critical level has been described by Page and Van Slyke,[24] and it can easily be used in office practice.

The main attention in therapy, therefore, should be directed toward attempting to raise the low plasma protein by the only means known, that is, by increasing the protein intake in the diet. From 150 to 200 grams of protein a day should be given and maintained for prolonged periods of time. Such a diet will usually be tolerated by the patient without evidence of nitrogen retention. As a precaution, however, the nonprotein nitrogen or urea nitrogen of the blood should be checked frequently, and if abnormal rises of these occur, the amount of protein in the diet should be reduced.

Although the low plasma protein level is the primary cause of nephritic edema, other factors which affect the water balance of the body must be considered, for it is often possible to influence the amount of fluid retained by altering or controlling such factors. Under all circumstances, both normal and pathologic, the water that is retained in the tissue spaces, and which,

if in great excess, is evident as edema, is present as salt solution in equilibrium with the salt content of the blood plasma. Sodium, in combination with chloride especially, is the important base that is concerned. Potassium, present in much smaller quantities, is chiefly held in the body cells. In the edema of nephritis, much sodium is retained in the body in the tissue fluids. This retention is not due to any impaired ability of the kidney to excrete it, but is merely a secondary effect from the low osmotic pressure, due to the reduced plasma proteins, which tends to hold water in the tissue spaces and, hence, sodium along with it. It has been shown that conditions which tend to reduce the sodium of the tissue fluids also tend to draw out the water, produce a diuresis, and so relieve the edema. Such conditions are: reducing the sodium or salt intake in the diet and using an acid-ash diet; giving acid-producing salts, such as ammonium or calcium chloride, in doses up to from 10 to 15 grams daily by mouth; and giving potassium salts instead of sodium. The mechanism by which the first operates is self-evident; if the sodium intake is restricted, while a certain amount is inevitably being washed out in the urine, the amount of sodium in the body will decrease. The giving of acid salts tends to produce loss of sodium since these salts are converted in the body into hydrochloric acid, which is combined to some extent with the sodium of the body and excreted as such. Finally, it has been claimed that the administration of potassium salts may tend to increase the excretion of sodium and, hence, to produce a diuresis. This contention, however, has been disputed.[25]

In patients with renal edema, therefore, the first attempt should be to increase the plasma proteins by a high protein diet. Attempts at regulating the edema by reducing the sodium of the body should also be instituted. This can be done by any or all of the three methods mentioned above. A word of caution regarding the last two of these regimes is in order. Particularly in cases with marked renal impairment, vigorous or prolonged administration of either acid or potassium salts may result in acidosis or potassium poisoning. Such therapy should be carried out, therefore, only for short periods and in cases without nitrogen retention.

Since water can be retained in the body only as salt solution, even fairly large amounts of water by mouth will not produce or aggravate edema, if sodium salts are not available. There is, therefore, no necessity for limiting the fluid intake of patients with renal edema, provided the salt intake is restricted.

In patients in whom the edema persists in spite of applying the methods above and in whom there is little evidence of severe inflammatory changes in the kidneys as judged by the absence of hematuria, it is proper to try the use of a diuretic. The most effective of these drugs are probably the mercurial preparations, such as mersalyl (salyrgan), merbaphen (novasurol) and mercupurin. Mersalyl should be given intravenously at 3 or 4 day intervals, starting with a dosage of 0.5 cc. and gradually working up to 1.5 or 2.0 cc. if no evidence of renal irritation occurs. It has recently been shown[28] that the administration of mercury by rectum in the form of suppositories (mercurin) is both feasible and effective, and this method of administration may come to be employed to a much greater extent in the future. Urea may also be tried as a diuretic, in amounts of from 45 to 90 grams daily. The effectiveness of diuretics in renal edema is usually much less than in edema of cardiac origin.

Renal Failure. In some cases of chronic glomerulonephritis, of malignant hypertension and of chronic bilateral pyelonephritis, the functioning kidney tissue may be reduced to such a point that renal failure supervenes, and eventually uremia may occur. The stages in this process, which may take place fairly rapidly or very slowly, can be summarized as follows:

1. The kidney loses its power to concentrate the urine. More water must be excreted to wash out the solid waste products and, hence, polyuria and nocturia occur.

2. A stage is reached when the nitrogenous substances of the blood cannot be maintained at a normal level even by this method, and nitrogen retention occurs. This may be rapidly progressive or remain comparatively stationary for weeks or months.

3. Certain conditions result which tend to produce acidosis and dehydration. (a) Inorganic acids, especially phosphates and sulfates, can no longer be adequately excreted and pile up in the body. (b) Chlorides are swept out with the dilute urine, and as the kidney further loses its power to manufacture ammonia, more and more fixed base, particularly sodium, is lost with the chlorides. This is aggravated by deficient salt intake due to anorexia, nausea or loss through vomiting. In the final stages the power to excrete an acid urine is lost, and the last defence against acidosis is gone.

4. The blood calcium may become lowered, which condition is probably secondary to the low plasma proteins and retention of phosphates.

5. Anemia of a greater or less degree may occur, primarily on account of a toxic depression of blood formation, although in many cases a deficient diet has contributed to its occurrence.

The treatment for renal failure is in many ways diametrically opposite from that for nephritis with edema. Since nitrogen retention and marked edema rarely occur together, the indi-

cations for therapy are not likely to conflict. The principles of treatment are as follows:

1. When nitrogen retention begins to occur, as judged by an elevated blood nonprotein nitrogen, the protein of the diet should be restricted. If this nitrogen retention is a slow process, there is no point in limiting the protein to less than the minimal nitrogen requirement of the individual,—probably about 40 to 60 grams,—as the patient will merely burn his own body protein.

2. Chlorides must be provided to take care of the loss in the urine. Ordinarily, 5 to 7 grams of salt added to what is normally present in a salt-poor diet will suffice.[27] In patients who are vomiting, it should be supplied parenterally.

3. Fluids should be given by mouth or parenterally in sufficient quantity to ensure, if possible, an output of 2,000 cc. daily in order to carry out as much waste substance as possible.

4. In the severe cases with acidosis, small amounts of sodium bicarbonate (2 to 3 grams daily) may help. Caution should be exercised in the amount given, however, because of the inability of such patients to handle alkalis well. Alkalosis can be precipitated by an overdosage of sodium bicarbonate.

5. If the symptoms of tetany occur or the blood calcium is very low, small doses of calcium may be given.

6. Attempts to relieve the anemia by iron or liver extract are usually unavailing, unless the anemia has been caused by blood loss or by a deficient diet. Transfusions may give temporary improvement.

Such treatment will often contribute much to the comfort of the patient and may prolong his life. In the cases, unfortunately not common, where the renal failure occurs as an acute exacerbation of the disease or where it is precipitated by some extraneous cause, such as heart failure or an infection, improvement may occur, and the patient may regain a degree of health for a considerable length of time.

SUMMARY

Various forms of Bright's disease are discussed in regard to etiology and treatment. These include: acute Bright's disease; urinary infections, both in the acute and end stages; and chronic Bright's disease in the latent stage, in the stage with renal edema and in the stage with renal failure (uremia).

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NEW ENGLAND SURGICAL SOCIETY

FURTHER REPORT ON OSTEOMYELITIS AT THE MASSACHUSETTS GENERAL HOSPITAL*

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TWO years ago one of us[11] presented before this society a brief consideration of the subject of osteomyelitis, together with a description of the work of a new Osteomyelitis Clinic at the Massachusetts General Hospital. At that time it was stated that a report of progress would be rendered in a year. A year was far too short a time, however, and even the 2 years that have already elapsed are not sufficient to enable us to bring back to you anything of outstanding importance or merit. The end result in cases of osteomyelitis is usually not a matter of weeks or months but of several or many years. It will, however, be worth while to discuss the problems that have presented themselves to us, although our ideas concerning them are far from crystallized.

Our clinic was organized under the leadership of one general and one orthopedic surgeon with the assistance of a few younger men. The patients have had more study and consideration than has ever before been their lot. Well-attended ward rounds have led to intensive thought concerning, and active discussion of, each case, while the frequent presence of visitors has supplied us with many helpful suggestions. The ward rounds occur once a week, an outpatient clinic is held once a week, and the ward cases are constantly in the hands of an interested member of the clinic.

A scrutiny of the literature reveals nothing that is strikingly new. Further studies on the bacteriology and immunology of the disease are being carried out in many clinics as well as in our own, and through these studies much help will probably come.[16] Each of the various methods for treating chronic osteomyelitis, such as the Baer,[4] the Orr[12] and the Stewart,[14] has its advocates and its good points, but no one of them solves the whole problem. The use of vitamins[2, 3] internally, and externally by means of cod-liver oil ointments, has attracted much favorable attention, and in our hands has been helpful but not a panacea. Furthermore, the mechanical problem of closing a big hole with devitalized bone at the bottom is always intriguing, and suggestions, such as that of Bozsan,[5] to bore and saw through the dead bone into the sound bone, in order to restore circulation, have been given consideration.

Of our 67 cases of disease of the long bones, only 7 were instances of acute osteomyelitis and 4 of subacute osteomyelitis (table 1), so that we had very little opportunity to treat such patients until the infection had passed beyond the early stages and had assumed the characteristics of a chronic inflammation. In one instance of an acute exacerbation of a chronic osteomyelitis of the femur with streptococcus septicemia, we believe that we saved the patient's life by immunotransfusions, as suggested by Lyons[10] of our clinic. We have had no experience with bacteriophage.[1] In acute osteomyelitis with staphylococcus septicemia, in which the signs and symptoms are due to toxemia, we believe that careful use of an antitoxin[9] may prove to be of value, and further observations on this problem will be carried out. Our tendency is to become more and more conservative in the handling of acute cases; we attack the local lesion only after the patient's condition has been made as favorable as possible, and then make the local procedure speedy and nonmutilating. An examination of the most recent book on osteomyelitis by Wilensky[17] reveals a valuable study of the subject with the concrete advice that, in operating on the local lesion in acute cases, nothing more radical should be done than to make drill holes in the bone, and that even this may be too much. A recent article by Humphries[8] states that operation in acute cases should be abandoned since, in many instances, such a procedure is positively harmful—to this we do not subscribe. The excellent article by Green and Shannon,[6] calling attention to the value of the utmost conservatism — even to refusing operation — in infants under 2 in whom the infection is due to the streptococcus, is a very valuable contribution.

TABLE 1. *Classification of Cases of Osteomyelitis Occurring in the Long Bones.*

	Acute	Subacute	Chronic
Femur	6	3	14
Multiple	0	0	18
Tibia	1	1	16
Humerus	0	0	8
Total	7	4	56

Our own problem has been chiefly the treatment of chronic osteomyelitis. Table 2 gives

*Read at the annual meeting of the New England Surgical Society at Bridgeport, Connecticut, September 26, 1936.

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the location of the lesions. There were, in addition, 13 that involved the jaw and 3 that involved the toes, which were not treated in the clinic. The femur, as is usual, showed the greatest frequency of involvement.

TABLE 2. *Bones Involved in Cases of Osteomyelitis Seen in the Clinic from October, 1934, to June, 1936.*

Femur	23
Multiple	18
Tibia	18
Humerus	8
Foot	7
Pelvis	5
Jaw	4
Fibula	2
Scapula	2
Metacarpal	2
Radius	1
Total	90

Deaths..... 2

Mortality = 2.2%

FIGURE 1.

Atypical acute osteomyelitis of femur, resulting in amputation.

The most discouraging cases are those that involve the femur (table 3). Of these 23 patients, only 5 are completely healed and 12 are being followed. Two acute cases that occurred in adults required amputation because of the uncontrollable spread of the disease from the lower end of the femur upward (fig. 1), and another amputation was required in a young boy who, after more than a year in the hospital, was slowly losing ground as a result of extensive infection and the resultant absorption. One death was due to a very atypical acute osteomyelitis of the upper end of the femur in a boy of 18, who died of septicemia in spite of every attempt to secure conservative drainage and to support his general condition (fig. 2). The second death was in a very young child who was moribund on entrance and lived only a few hours. The chronic cases have presented the problem of closing large cavities in which there is little tendency for the gutter in the bone to fill in. We have handled this by various methods of local treatment, chiefly by keeping the external portion of the wound packed widely open for long periods of time after the cavity in the bone has been completely unroofed. No particular kind of antiseptic has been especially effective.

TABLE 3. *Results of Treatment in Cases with Osteomyelitis of the Femur.*

Good, with complete healing	5
Incomplete healing	12
Amputation, to save life	3
Dead	2
Lost	1
Total	23

FIGURE 2.

Atypical acute osteomyelitis of upper end of femur, resulting in death from septicemia.

Table 4 gives the results of treatment in 18 cases of multiple osteomyelitis. In most instances each patient had a subacute exacerbation

in some previously drained area of bone infection. These patients were not seriously sick but required local drainage and postoperative treatment.

Osteomyelitis of the tibia (table 5) is less discouraging than that of the femur because, unlike the latter, the infection occurs in a superficial bone which is easy of access and is not covered with thick, soft parts that tend to fall in and close over the draining bone. In two instances, cancer developed in very old, neglected cases; the patients had come to us after having had the disease many years. These cases required amputation, but proper treatment in earlier years might have prevented this. Our only

TABLE 4. *Results of Treatment in Cases with Multiple Osteomyelitis.*

1. Good, with complete healing	6
2. Incomplete healing	10
3. Lost	2
Total	18

TABLE 5. *Results of Treatment in Cases with Osteomyelitis of the Tibia.*

1. Good, with complete healing	10
2. Incomplete healing	4
3. Amputation for cancer	2
4. Lost	2
Total	18

slide skin graft was performed in one of these tibial cases to protect an area of tibia that was covered only by a paper-thin sheet of new skin which was being continually knocked off (fig. 3).

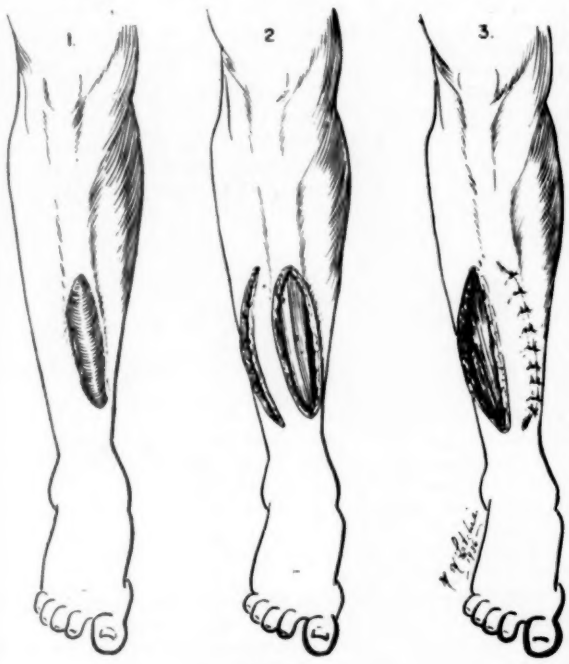

FIGURE 3.

Slide graft to cover bare area over tibia, following osteomyelitis.

In 2 cases with small superficial cavities in the tibia, the roof was removed, together with the overlying soft parts, and no packing was inserted. In each of these cases, the cavity filled with blood clot and closed in rapidly from the bottom, so that the results were excellent.

The cases of osteomyelitis of the humerus (table 6) were not striking and healed well. Six

TABLE 6. *Results of Treatment in Cases with Osteomyelitis of the Humerus.*

1. Good, with complete healing	6
2. Incomplete healing	2
Total	8

of the 8 patients were entirely cured, 1 of the others was nearly healed, and in only one instance was the result unsatisfactory. This last case was one of atypical involvement of the upper end of the humerus, with secondary infection of the structures of the axilla; in fact, the axillary involvement overshadowed the bone disease, and it was thought that the infection in the axilla, rather than that in the bone, was responsible for the chronicity of the process.

TABLE 7. *Results of Treatment in Cases with Osteomyelitis of the Pelvic Bones.*

1. Good, with complete healing	2
2. Incomplete healing	2
3. Lost	1
Total	5

Table 7, showing the results of the 5 cases of osteomyelitis of the pelvic bones, does not cover several cases that have recently been observed, and this type of the disease is so important and difficult to treat that it will be made the subject of a separate communication. In these cases the ischium was involved once, the ischium and pubic bone once, and the ilium three times. It has been our experience in such cases that the only satisfactory method of treatment is a very radical surgical operation, with wide exposure and removal of all diseased bone followed by packing and frequent dressings. The course of the healing process is always long, but if the operation has been thorough, the result will be satisfactory.

The scattered cases that are included in table 2 will not be taken up in detail, as they do not add anything relevant to the discussion at hand.

To return to the treatment of the various types of osteomyelitis we will give our ideas briefly under two headings.

Acute Osteomyelitis

1. No local operation should be undertaken until the patient's condition warrants it.

2. Local operation should be conservative. The soft part or subperiosteal abscess should be drained and, if there is a definite suspicion of pus in the bone, a few drill holes may be made,

but no widespread removal of bone substance should be attempted. The so-called "saucerization" or "unroofing" of a bone cavity is best left until a later date. In young infants, we would certainly adhere to the conservative principles advocated by Green and Shannon.[6]

3. In cases with a streptococcus septicemia, immunotransfusions should be done, and in those with a staphylococcus septicemia and toxemia, an antitoxin may be tried.

Chronic Osteomyelitis

1. Any bone cavity containing pus should be thoroughly uncovered. Following this, one of two methods may be carried out.

a. The wound may be thoroughly packed. The packing should be changed every few days, if necessary under an anesthetic. The external wound must be kept wide open by occasional revision or by cutting away the soft parts, again under an anesthetic if necessary. A constant drip of Dakin's solution may be used if the wound is dirty, although this has no marked effect on the healing. In our hands, the Stewart treatment, consisting of the use in the wound of a solution of picric acid and glycerin, which is followed by calcium carbonate, has been helpful in cleaning and stimulating certain dirty cavities. Cod-liver oil ointment should be given a further trial in order to evaluate more extensively its effect.

b. The wound may be closed with glass cannulas (devised by Smith-Petersen) sewed in at each end. A constant stream of Dakin's solution should be kept running through the depths of the wound. This has, in several instances, resulted in satisfactory healing and has done away with the usual process of prolonged packing and slow closure.

CONCLUSIONS

1. Acute osteomyelitis should be treated with due regard to the patient's condition, and the consideration of a local operation should be made consequent to this.

2. Chronic osteomyelitis should be treated by thorough, even radical, exposure of cavities and removal of sequestra. No one type of dressing is adapted to all cases; each must be treated according to the individual indications.

3. The future solution of the satisfactory treatment of acute and recurring osteomyelitis will come with increasing knowledge of bacteriology and immunology.

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DISCUSSION

DR. EZRA JONES, Manchester, N. H.: I think that this paper is remarkable for two features: first, that the author has not tried to give a panacea for osteomyelitis; and, secondly, that he suggests that the proper way to attack this problem is from the side of bacteriology and immunology.

In fact, his paper is a very comforting one. I make these observations from experience with 28 cases of osteomyelitis in the last 2 years. Twelve were in children, and 10 of these were acute. The other 16 were in adults, 6 being acute cases. Treatment in every instance has been by incision and drainage. My experience does not coincide with that of Humphrey, who believes operations are harmful, because I have seen some very spectacular relief result from opening the medullary canal.

In the acute cases as little operative work as possible was done. In every instance a small portion of the cortex was removed. The cavity was packed immediately with vaseline gauze. This gauze was irradiated at times, but I am not convinced that the irradiation had any effect. The gauze was left in from 4 to 5 weeks, when possible, as we believed that frequent dressings were detrimental. In practically every case a snugly fitting plaster with a window over the incision was applied.

It seems that the cases that were attacked early, before there was any evidence of bone destruction in the x-rays, did much better and were less apt to sequestrate than those that were treated later.

Autogenous vaccine has been used in children, and we feel that it has prevented multiple lesions. A difference has been noted between the streptococcus and the staphylococcus cases. Patients with streptococcus infection are much sicker, but sequestration is much less frequent.

It has been noticed that secondary lesions are much more tractable than the initial lesions and sequestrate less frequently. This probably indicates that the patient has developed an immunity, and is a rather convincing argument that a consideration of such a state is probably the most important way of attacking the problem.

NEUROLOGIC COMPLICATIONS FOLLOWING THE ADMINISTRATION OF VACCINES AND SERUMS*

Report of a Case of Peripheral Paralysis Following the Injection of Typhoid Vaccine

BY LEON J. ROBINSON, M.D.†

THE occurrence of a case of peripheral nerve paralysis following the prophylactic administration of typhoid-paratyphoid A and B vaccine is here recorded. This case served to emphasize the possibility of this rather rare complication. It also led to a consideration of similar neurologic complications which are occasionally encountered in the administration of serums, antitoxins and vaccines.

CASE REPORT

The patient, a 33 year old white male, has been a patient at the Monson State Hospital since August 4, 1935. The admission diagnosis was epilepsy, symptomatic of alcoholism. The family history was essentially negative and the past history negative except for occasional alcoholism.

Physical examination on admission was entirely within normal limits. On January 23, 1936, the patient was given 0.5 cc. of typhoid-paratyphoid A and B vaccine. This was administered subcutaneously as part of the routine prophylaxis against typhoid fever that is given to all patients and employees under 50 years of age in state institutions in Massachusetts. This same bacterial vaccine‡ from the Massachusetts Antitoxin and Vaccine Laboratory has been regularly used for this purpose without mishap.

Following this first injection into the left deltoid area, no ill effects were noted, and on January 31, 1936, the second injection of 1 cc. was administered into the right deltoid area.

On February 4, 1936, 4 days after the second injection, left foot drop was noted. The patient was able to give a reliable history and denied shooting pain, paresthesias, numbness or antecedent trauma. The attendant on the ward also supported this history of apparent spontaneous foot drop.

Physical examination at this time can be summarized as follows: The pupils were equal and reacted to light, although the left was slightly less active than the right. Both were regular and reacted well to accommodation. There was no nystagmus, lid lag, diplopia or hemianopsia. The fundi were normal, although at first the optic disks were thought to appear slightly pale. Later examination found them normal in appearance. The nose and throat were normal. The teeth were in fair condition. There was no facial or frontalis paralysis. Bone and air conduction were normal, and the ear drums clear and intact. The hearing was normal. The heart showed no enlargement, murmurs or irregularities. The second pulmonic sound was louder than the second aortic sound. The blood pressure was 120/70, rising but 2 mm. in systolic and diastolic pressure when the left hand was immersed in cold water, that is, a negative test for latent hypertension. The lungs were clear and resonant throughout. No masses, tenderness, spasm or hernias were found on abdominal examination. The abdominal reflexes were absent. The cremasteric reflexes were sluggish, especially on the left. The knee jerks and the triceps, biceps and radial reflexes were equal and active bilaterally. The Hoffmann, Babinski, Chaddock, Oppenheim, Gordon, Kernig and Romberg signs were absent. There was no ankle clonus or past-pointing; no tremor of the extended arms and hands; no tremor or deviation of the tongue. The left ankle jerk was less active than the right. There was no tenderness over any of the peripheral nerves, and no adenopathy.

There was left foot drop with total inability to dorsiflex the foot. With the left heel on the floor the patient could not raise the ball of the left foot from the floor. There was also marked weakness on plantar extension of the foot. The left leg was weaker than the right on extension and flexion, at the knee, against resistance. Abduction of the left leg at the hip was unimpaired. Sensation to pain and to vibration in the lower extremities was normal and equal. At this time no atrophy of the left lower extremity was noted. The patient was afebrile throughout the period of observation.

Laboratory Findings. Lumbar puncture revealed an initial pressure of 150 mm. of water with normal dynamics on jugular and abdominal compression. On removal of 15 cc. of clear, colorless fluid the final pressure was 100 mm. No cells were found; the globulin was 0, the chlorides 730 mg. per 100 cc., the sugar 77 mg. per 100 cc., and the total protein 21 mg. per 100 cc. The colloidal gold curve was normal and the spinal fluid Wassermann was negative.

The red blood cell count was 4,590,000; the hemoglobin 76 per cent (Sahli); the color index 0.8; the initial white cell count 8,300 with polymorphonuclears 61 per cent, large lymphocytes 1 per cent, small lymphocytes 34 per cent, transitionals 3 per cent, and basophils 1 per cent. The red cells were reported normal in appearance. The white cell count one week later was 8,800, with polymorphonuclears 49 per cent, small lymphocytes 44 per cent, transitionals 5 per cent, basophils 1 per cent and eosinophils 1 per cent. The Hinton test on the blood serum was negative. Urinalyses were negative. The blood sugar tolerance test showed a fasting content of 100 mg. per 100 cc.; ½ hour after the glucose 150 mg.; 1 hour 130 mg.; 2 hours 110 mg.; 3 hours 80 mg. The blood cholesterol content (fasting) was 140 mg. per 100 cc.

X-rays of both radii failed to reveal any epiphyseal or metaphyseal bone pathology suggestive of lead poisoning.

Course and Treatment. The patient at first received infrared light treatment and passive motion with gentle massage. Later, active motion and diathermy were added.

On March 4, 1936, 1 month after the onset of the foot drop, there was no recovery of motion in the muscles involved. With the left heel on the floor, the patient was still unable to raise the ball of the foot from the floor. At this time there was obvious atrophy of the left leg, the left ankle and calf being 2.0 cm. less in circumference than the corresponding circumferences on the right.

On April 21, 1936, 2½ months after the onset of

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‡Contains 0.4 per cent cresol as preservative.

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the foot drop, the absolute foot drop and the atrophy were still unaltered. A partial reaction of degeneration was elicited in testing the left leg. The remainder of the physical examination was not remarkable. By this time interrupted negative galvanism had been added to the therapy of the left leg paralysis without any noticeable improvement in the condition. On May 1, however, the patient was able to raise the ball of the foot 1.5 cm. from the floor with the heel held to the floor. This was almost 3 months after the onset of the palsy.

On May 19, 1936, 3½ months after onset, practically complete recovery from the peripheral paralysis was noted. With the left heel touching the floor the patient was able to dorsiflex the left ball of the foot 10 cm. from the floor, as compared with dorsiflexion of 12.5 cm. in this position with his normal right foot. There was as full and as strong plantar extension of the left foot as there was of the right. The circumferences of the left ankle and left calf were but 0.6 cm. less than the right; the measurements were: right calf 35 cm., left calf 34.4 cm., right ankle 26.9 cm., left ankle 26.3 cm.

Diagnosis. A diagnosis of peripheral paralysis complicating typhoid vaccination was made.

Anterior poliomyelitis was ruled out by the absence of a febrile course, absence of muscle pains, and negative spinal fluid findings. Alcoholism, as the cause of a peripheral neuritis with palsy, was ruled out by the absence of recent long-continued bouts of alcoholism and of tenderness over the peripheral nerves. Lead palsy was ruled out by the absence of bilateral nerve involvement, of a lead line in the gums, of stippling of the red blood cells and of a lead line in the radii. Avitaminosis was ruled out by the presence of an adequate diet and the absence of skin manifestations, of bleeding gums, of sore tongue and of gastrointestinal symptomatology. Diabetic neuritis with palsy was ruled out by the absence of tenderness over peripheral nerves and of pain and by the presence of a normal blood sugar tolerance test.

REVIEW OF SIMILAR CASES

The use of serums and vaccines prophylactically and therapeutically is becoming universally more common. The physician who uses them is generally well aware of the possibility of such obvious complications as local infections at the site of injection, foreign protein reactions, and the introduction of *Treponema pallidum* and the tetanus bacillus. But he seldom has occasion to consider the nervous system complications that rarely follow the parenteral introduction of vaccines and serums.

It is well known that the central nervous system is extremely susceptible to a number of diverse noxious materials and conditions.

A list of some of these central nervous system noxae that have been parenterally introduced and the conditions that they have caused include: neoarsphenamine during antisyphilitic therapy causing sympathicoplegia;[1] intravenous evipan for general anesthesia causing flaccid paralysis of the extremities;[2] cocaine derivatives injected into the subarachnoid space for spinal anesthesia causing neurologic complications of the most varied type;[3] quinine bismuth iodide during therapy for neurosyphilis causing paralysis;[4] emetine hydrochloride injections for amebiasis causing paresis;[5] gold salt therapy for pulmonary tuberculosis causing fatal Landry's paralysis,[6] and so forth. The frequency of peripheral and central nervous system complications following the administration of rabies vaccine is common knowledge, and the literature contains many such reports.[7]

In recent years there have been reported similar, uncommon, but by no means insignificant, complications following the administration of other vaccines and of serums. Among the offenders are every type of serum and vaccine, including tetanus antitoxin,[8, 9, 20] diphtheria antitoxin,[9, 10] scarlet fever antitoxin,[11, 20] scarlet fever toxin for prophylaxis,[12, 13] antipneumococcic serum,[14] serum for tuberculosis,[19] antimeningococcic serum,[15] staphylococcus vaccine,[16] typhoid vaccine[12, 17] and smallpox vaccine.[18]

In reporting a case of axillary nerve paralysis following the prophylactic injection of tetanus antitoxin, Young[19] recorded from the literature 47 cases of neurologic complications following serum, and only 3 following vaccine.

Doyle,[20] reporting 2 cases of flaccid brachial paralysis following scarlet fever and tetanus antitoxin, respectively, collected 47 similar cases from the literature. Doyle purposely limited his report to cases following serum administration and omitted all cases due to vaccines. Of his 47 collected cases, 8 had been included in Young's previous review.[19]

CLINICAL PICTURE

The usual sequence of events, whether serums or vaccines are administered, is the gradual or sudden onset of a flaccid paralysis of the lower motor neuron type, occurring at any time from the same day to 60 days after inoculation (usually occurs within 14 days) and involving any peripheral or even cranial nerve or nerves (brachial, ulnar, radial, axillary, sciatic, long thoracic, lumbar, or even facial, optic[12, 14] or auditory nerves).[8e] Less often the spinal cord or higher centers are involved. Serum sickness and urticaria may or may not accompany or precede the paralysis. Urticaria, with subsequent nerve involvement, has even followed typhoid[12] and rabies vaccine[7n] injections. Atrophy is a frequent but not an invariable feature, and may be accompanied by the reaction of degeneration. Atrophy generally occurs within 2 days to 3 months but has been known to occur as long as 7 months after inoculation.[19]

Residual paralysis persisted in 48 per cent of Young's 37 collected cases with peripheral nerve involvement, whose outcome was known.[19] As many as 21 per cent showed no recovery at all, while 27 per cent showed only partial recovery. The other half of the cases recovered completely.

Doyle, on the other hand, found a more favorable group of cases and stated that "most

of the cases recover completely.''[20] However in 25 of his collected cases, including 1 of his own, no mention is even made as to end results. In cases where improvement was mentioned it occurred at any time from 10 days to 18 months after the onset and was not always complete.

The prognosis, while guarded as to recovery in peripheral involvement, is definitely serious as to life in central nervous system involvement, which carries with it a mortality of over 50 per cent.

The central nervous system type of complication may involve an anaphylactic response with nuchal rigidity, convulsions, coma, increase in spinal fluid cells, and death. This form generally occurred in cases in which intraspinal serum therapy was administered as in meningococcic meningitis or tetanus.[19]

Another form of central nervous system involvement is characterized by bizarre manifestations, which may involve any part of the central nervous mechanism. Thus, 4 days following scarlet fever toxin injected for prophylaxis Kennedy's case[12] developed urticaria, and 3 days later aphasia and partial right hemiplegia, followed 5 days later by right homonomous hemianopsia and bilateral swelling of the optic disks with retinal edema. Lumbar puncture revealed only increased pressure and 14 lymphocytes per cubic millimeter of fluid. Recovery occurred in 4½ weeks.

Another of Kennedy's cases received typhoid vaccine. Fever and urticaria followed the second inoculation. Twenty-four hours after the third and last inoculation, numbness appeared in the feet and spread to the waist. Four days later numbness was present in the ring and little fingers of both hands. There were incomplete glove and stocking anesthesia, total loss of vibration sense in the legs, tingling in the hands and feet, and weak and awkward grasping power. Deep reflexes were present but reduced, and the abdominal reflexes were absent for a month. Early in the disease there was difficulty in focusing near objects in the midline. Gradual general improvement occurred.

Hemiplegia has been reported following tetanus antitoxin.[21] In the central nervous system type of complication, lumbar puncture may reveal normal findings or such alterations as lymphocytosis[12, 8f], increased pressure,[12] increased albumin,[8f] all of these together[14] or marked pleocytosis.[19]

Subsequent to typhoid vaccination there have occurred Landry's paralysis,[22a] acute myelitis,[22b] hemiplegia and hemianopsia, epileptic crises, meningeal accidents, and polyneuritis,[17a] as well as asthenia, weakness and depression.[22c]

Three clinical forms of central nervous system complications subsequent to rabies vaccination are well described by Bassoe and Grinker.[7r] These are: the acute ascending paralysis of Landry's type; dorsolumbar myelitis; and neuritic forms, in which after a brief febrile period, one or more peripheral nerves become involved.

Following rabies vaccination there have been reported fatal myelitis,[7o] encephalomyelitis[7p, 7r, 7v], and extrapyramidal disturbance of gait with myoclonus.[7q]

Thus it will be noted that the administration of almost any serum or vaccine may be followed by an unusual complication of a neurologic nature.

DIFFERENTIAL DIAGNOSIS

The peripheral nerve complication subsequent to serum or vaccine injection is often difficult to diagnose as a complication of the inoculation, and when the complication occurs more than 2 weeks after the injection, confusion is especially apt to arise. It is to be distinguished from anterior poliomyelitis (which was the erroneous diagnosis in one of Kennedy's cases[12]), peripheral paralysis due to lead poisoning, neurologic complications such as occasionally occur in diabetes,[23] and the peripheral paralyses caused by alcoholism or avitaminosis. The central nervous system complication may simulate any disease of the cord or higher centers.

ETIOLOGIC MECHANISM

The exact etiology of these neurologic complications is still unknown. It is obvious from the frequency with which the palsies occur in localities other than the extremity into which the injections have been made that the inoculation is not the cause of a local injury to nerve tissue.

There are several mechanisms suggested to account for these neurologic complications. In the instances where horse serum has been the offending substance, the mechanism that has been invoked is perineural edema occurring locally as part of a general urticarial response to the serum.[8b, 9, 12, 20, 24] The nerve damage is assumed to be caused by pressure on the blood vessels of the nerve with resulting ischemia. Young denies that this explanation holds for every case. He points out that in a number of cases paralysis developed without any previous serum disease and that in still other cases paralysis came on from 10 to 15 days after subsidence of the serum reaction.[19]

The action of a specific neurotoxin has been favored, rather than anaphylaxis, in 2 cases in which no previous serums had been given to cause sensitization.[8f] Urticaria followed the administration of tetanus antitoxin in both of these. Gordon favors a specific neurotoxin in the serum plus ''shock'' from the introduction of a foreign protein.[13]

Some cases following the injection of rabies vaccine have been attributed to a foreign protein reaction,[36] but the condition has generally

been said to be an attenuated rabies virus disease transmitted by the vaccine.[7r, 7u]

To substantiate this claim, the proponents point to the similarity of perivascular softening of the spinal cord in a case of Landry's paralysis following antirabic vaccination with the changes found in subacute rabies.[7r] Stuart and Krikorian[7n, 7s] decided that the cause of these complications was a cytotoxin in the basic nerve substance of all antirabic vaccines, which affected peculiarly susceptible persons, rather than the cause being the phenol preservative or a virus. In 1908 the cytotoxin theory was also favored by Marinesco[25] and in 1932 by McKendrick.[7w] Marque[7t] blames the virus contained in the antirabic vaccine, enhanced by the rapid administration of the vaccine.

In attempting to attribute to a common cause the etiology of neurologic complications following the injection of so many different substances, one can consider several possibilities common to all of these injections. These are a chance virus contamination, the foreign protein or the preservative added to the material.

If the chance introduction of a virus were at fault there would doubtless be similar neurologic complications following subcutaneous injections of such drugs as caffeine, morphine, adrenalin and other medicinals administered subcutaneously.

All serums and vaccines contain foreign protein whether in the form of animal serum or the protein of the dead bacteria in bacterial vaccines and of the spinal cord emulsion in rabies vaccine. We have already quoted the opinions favoring a foreign protein reaction. Yet cases have been cited in which no previous sensitization had preceded the etiologic inoculation.[19]

Finally it should be considered whether similar preservatives common to these vaccines and serums might be the offending agents in some instances. In the present state of uncertainty, even this possibility may deserve the following discussion. Phenol, or one of its compounds, is often used as a preservative in rabies vaccines, typhoid vaccines and animal serums.* It is conceivable that in susceptible individuals phenol in the material administered subcutaneously might cause neurologic complications.

The paralysis following the ingestion of Jamaica ginger was found to be caused by a phenolic compound, triorthocresyl phosphate.[27] Polyneuritis and paralysis following the ingestion of the abortifacient, apiol, have also been shown to be caused by this same phenolic compound.[28] Another phenolic compound, a cresol phosphate, has been the causative agent of polyneuritis in tuberculous patients to whom the drug had been given for therapy.[29]

Of phenol poisoning it is stated that "the systemic action of phenol is almost confined to its effect on the central nerve system; and there is hardly a portion of this which escapes its action . . . Cresols closely resemble phenol in their action—and, like it, also exert their chief systemic action on the central nerve system."[30] The symptoms of triorthocresyl phosphate and creosote poisoning are weakness, polyneuritis, paralysis and atrophy,[28d] and they are similar to the symptoms of the neurologic complications following the injection of serums and vaccines.

Against the "phenol hypothesis" many valid objections can be raised. It has been maintained that triorthocresyl phosphate alone, of all the phenolic compounds, is able to produce neurologic pathology and symptomatology.[31] To prove this point the investigators injected triorthocresyl phosphate subcutaneously in a large series of animals (7 dogs, 11 monkeys and 9 fowl) and produced paralyses in them. Then to a small group of animals (2 dogs, 1 monkey and 1 fowl) they administered phenol orally. The only monkey to which phenol was administered developed acute fatal phenol poisoning. At autopsy this monkey "showed a slight involvement of the peripheral nerves and distinct changes in the anterior horn cells." These pathologic changes are strikingly similar to those produced by triorthocresyl phosphate in the same observers' series. It seems strange that the group given phenol was not only so small, but also that the oral route was used rather than the subcutaneous route of administration as in animals given triorthocresyl phosphate. Furthermore, the phenol was administered in an aqueous or alcoholic solution. In the tables published by these investigators, it is noted that, in all their cats given aqueous or alcoholic solutions of triorthocresyl phosphate orally, no definite paralysis occurred. Oral administration of triorthocresyl phosphate caused paralysis in their cats only when given in an emulsion of acacia, in which form phenol was not similarly administered.

It is known that orthocresol is definitely more toxic than phenol.[32] But the more frequent occurrence of neurologic complications following the administration of triorthocresyl phosphate as compared with their rarity following phenol-containing substances does not exclude phenol as a definitely possible though rarer cause of these complications.

The rarity of neurologic complications following phenolized serums and vaccines may be construed as evidence against phenol as an etiologic factor because of the frequency of such inoculations. And yet there are differences in susceptibility not only in different individuals but in the same individuals at different times. Many

*Phenol or tricresol (0.2-0.5 per cent) is the preservative in many producers' rabies vaccine (of the Cutter Laboratory, Berkeley, Calif.; Eli Lilly Company, Indianapolis; Gilliland Laboratories, Marietta, Pa.; Hixson Laboratories, Johnstown, Ohio; Lederle Laboratories, New York; Sharp and Dohme, Philadelphia; E. R. Squibb and Sons, New Brunswick, N. J.; and many others too numerous to mention); in typhoid vaccine (of Eli Lilly; of Antitoxin and Vaccine Laboratory of Massachusetts, and of many other commercial firms); and in numerous bacterial vaccines, serums and antitoxins.

of the cases, as in the case reported in this paper, showed no ill effects from the same offending substance when injections were performed previous to and subsequent to the injection that did initiate symptoms.[7r, 12, 17]

A more serious objection to the phenol theory is perhaps present in the claim that the neuroparalytic complications that are produced experimentally in a small percentage of rabbits and white rats by repeated inoculations of nerve substance can be prevented by exposure of the brain suspensions to the action of 1 per cent phenol with or without heat.[7s] Moreover, paralytic accidents have followed the administration of the desiccated and diluted rabies vaccine as well as the phenolized rabies vaccine.[7u] Finally, there is no one substance, which to the exclusion of all others, may be noxious to the nervous system. Neurologic complications have been evoked by many agents, such as neoarsphenamine, evipan, cocaine, quinine bismuth iodide, emetine and gold salts, none of which contains phenolic compounds.

In reporting cases of neurologic complications following vaccine or serum administration it would be of interest to record also the preservative in the vaccine or serum used.

PATHOLOGY

Unfortunately pathologic material is scanty and throws little light on the mechanism. No autopsy material is found in cases of peripheral nerve complications following serum. Only one autopsied case in which serum had been used could be found by Young as upholding the theory of perineural edema in the cases of involvement of the central nervous system with meningeal symptoms.[19]

Cases of rabies vaccine complications are richer in autopsy material. The pathology in 2 autopsied cases has been described as an intoxication in contradistinction to an inflammation, with the greatest damage being found in the ganglion cells, in which were observed slight chromatolysis to complete necrosis.[7n] In the same paper the observation is made that the pathology of similar cases in the literature varies greatly from hyperemia, congestion or edema of the brain, cord and investing membranes to a hemorrhagic myelitis.

The autopsy findings in a case of Landry's paralysis following inoculation with rabies vaccine in a patient not bitten by a dog have been reported. The perivascular softenings in the gray and white matter of the spinal cord and their similarity to the changes found in subacute rabies led the authors to believe that such cases had an attenuated rabies virus disease.[7r]

An interesting observation is reported of a German shepherd dog that was inoculated with antirabic vaccine and developed a fatal, generalized, flaccid paralysis resembling the ascending Landry's paralysis in man. Rabies was ruled out by the short incubation period of 10 days, absence of Negri bodies, and negative result on attempted animal transfer of rabies. Acute catarrhal gastroenteritis, lymphadenitis of the mesenteric nodes, and, in the central nervous system, meningeal hyperemia, cerebral and medullary edema, and acute internal hydrocephalus were found at autopsy.[26]

The nerve lesions produced experimentally in animals by triorthocresyl phosphate consist of degeneration of the myelin sheaths of the peripheral nerves with secondary degeneration of the axis cylinders.[28d] In addition to these lesions Smith and Lillie found degenerative changes in the anterior horn cells throughout the spinal cord in animals given triorthocresyl phosphate subcutaneously, and in 1 monkey given phenol orally.[31]

Jeter reported a case of a man with paralysis from Jamaica ginger (since Jeter's report proved to be caused by triorthocresyl phosphate) in which perineural exudate about the peripheral nerves was the only significant lesion.[33] Autopsy of 3 patients with Jamaica ginger paralysis but dying of other causes revealed myelin sheath and axis cylinder degeneration of the peripheral nerves.[34]

PROPHYLAXIS

There is no way of knowing what patients may be unexpectedly affected by this unusual complication. The physician should not be held responsible for its occurrence, and the possibility of its happening is not a contraindication to the intended injection.

TREATMENT

Rest during the early days is indicated for the peripheral paralysis as in any peripheral neuritis. This is to be followed by physiotherapy, which may consist of infrared light, gentle massage and passive motion followed later by active motion (both types of motion to bring into play the affected muscles and so prevent disuse atrophy), and still later interrupted negative galvanic current, which may be of benefit. The presence of a reaction of degeneration usually indicates a poor prognosis, although cases, like the one reported, have been recorded in which a partial or even complete reaction of degeneration has been followed by recovery—more often by partial recovery.[19]

Cases with more extensive neurologic involvement than simple peripheral paralysis carry a graver prognosis both as to life and as to return of function.

SUMMARY

A case of flaccid peripheral paralysis following the administration of typhoid-paratyphoid A and B vaccine is presented. A careful elimination was made of other possible causes for this paralysis, which was noted 4 days after the sec-

ond injection of the vaccine. At this time there occurred left foot drop, followed later by atrophy and reaction of degeneration. Physiotherapy was administered, and 3½ months after the onset recovery was practically complete and improvement was still progressing.

A review of similar neurologic complications following serums and vaccines is presented, and their clinical pictures are described. The differential diagnosis demands distinction from peripheral paralyses caused by anterior poliomyelitis, lead poisoning, diabetes, alcoholism and avitaminosis.

The etiologic mechanisms that have been invoked in the past include perineural edema resulting from a local manifestation of a general serum reaction, a specific neurotoxin and an attenuated virus disease.

In commenting on possible etiologic mechanisms, a consideration has been given to the phenolic compound so frequently used as a preservative in serums and vaccines. In this connection there is presented the evidence which incriminates triorthocresyl phosphate as the cause of peripheral neurologic involvement in apiol and Jamaica ginger poisoning. The similarity in both symptomatology and pathology in these cases, in cases following serums and vaccines and in a reported case following experimental phenol poisoning in a monkey is also pointed out.

Physiotherapy is advocated as the treatment of such peripheral neurologic complications as may occur following the administration of serums or vaccines.

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A REPORT ON SIX CASES OF SUPRACONDYLAR FRACTURES OF THE ELBOW

BY JOHN D. ADAMS, M.D.*

IT is not the writer's intention to present anything new or original in the treatment of supracondylar fractures of the elbow. The intelligent interpretation of one's own experience is still the basis on which one should improve his technic.

The average physician is apt to treat supracondylar fractures of the elbow on the assumption that if he places the elbow in acute flexion, he has accomplished a perfect reduction. He is guided also by the teaching that all fractures should be reduced immediately, irrespective of existing conditions. Nevertheless, he often disregards the amount of force used and the manner of its application during his manipulations. Fortunately, however, it is inconceivable that any physician, trained house officer or junior surgeon connected with a hospital could possibly reduce such a fracture and leave the patient without testing the radial pulse.

There is scarcely any other fracture of the human skeleton where lack of accurate knowledge of the anatomy of the part, together with a misconception of the relations between the fragments and the influence exerted by the position of the extension after reduction may contribute to such grave complications in unskilled hands. The old saying, "it is the first dressing that counts," is homely language but still holds true. It is what the first doctor who sees a fracture does, or does not do, that establishes the future function of that particular joint.

With the passage of years our results have improved, but, sad to relate, there are still too many deformed elbows. We are busy developing standards and "methods of doing," but do we emphasize enough the "don'ts"? Our experiences are only too full of unguarded prognoses, grave complications and end results that demonstrate an utter disregard of the correct application of surgical principles.

It has occurred to the writer that an analysis of six supracondylar fractures of the elbow that came into his hands in a period of 90 days might serve to illustrate some of these points.

Case 1. A boy, aged 6, fell a distance of 15 feet, striking his left elbow. He was taken to a nearby hospital where x-rays showed a typical supracondylar fracture of the humerus (fig. 1). The patient was anesthetized, and an attempt was immediately made to reduce the fracture, but without success. A second similar manipulation was performed 2 hours later. There was a considerable amount of force used in each attempt, and traction was applied with

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the arm in full extension. After the second attempt at reduction, the arm was forced into acute flexion at the elbow. The x-ray check-up at this point showed that the proximal fragment was still anterior. The radial pulse was absent. The arm was then dropped into full extension. The writer saw the patient about 6 hours after the injury. The examination at this time showed a tremendously swollen elbow, which was being held at approximately a right angle. The radial pulse was faint but palpable. The arm was put in full extension, and an icebag was applied to the elbow. The radial pulse returned to normal in about 12 hours and was watched for the

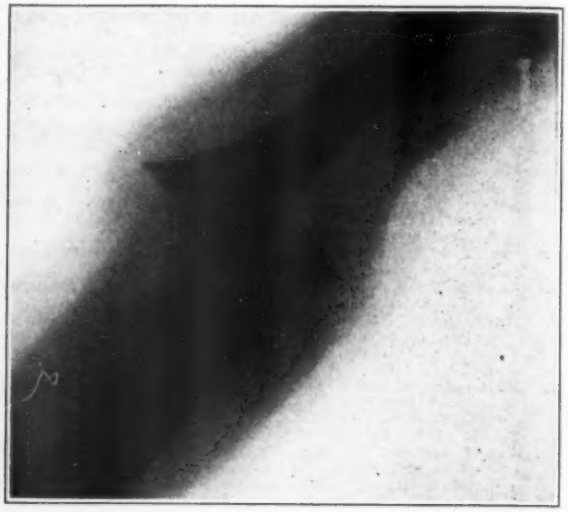
CASE 1. FIGURE 1.
Original fracture.

next few days. The arm was allowed to remain in full extension for a period of 5 days. At that time the patient was given ether anesthesia and, owing to the fact that a sharp spicule of the proximal fragment gave evidence of coming through the skin, the fragments were exposed through an internal lateral incision, and a fairly good-sized blood clot was removed from the joint. The fragments were then apposed, after which the arm came up, and it was possible to put the elbow into acute flexion with a palpable radial pulse. Four days later the boy had a temperature of 104° F. The following day, since the temperature was still elevated, the stitches

CASE 1. FIGURE 2.
End result 1 year later.

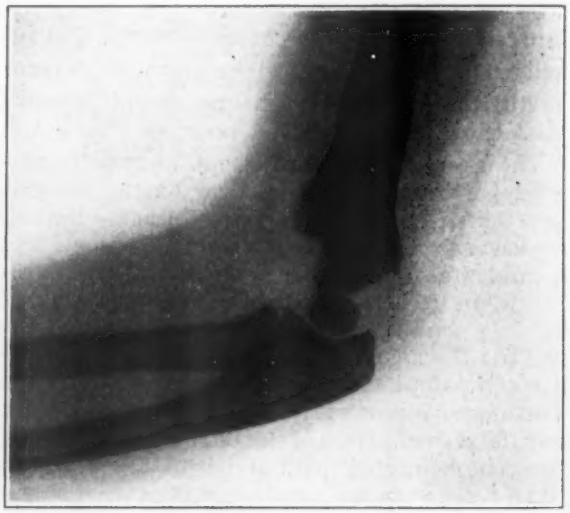
were removed, the wound was opened, and about ½ dram of pus was evacuated. No culture of the pus was made. Dressings were done daily for a period of 2 or 3 weeks. The temperature dropped to normal 3 days after the wound was opened. An x-ray check-up showed no bone involvement. On questioning the boy's mother, it was found that the boy had recovered from an attack of scarlet fever about 8 weeks prior to the injury. The wound healed completely in 6 months. A final check-up 14 months after the injury showed that the elbow went to within 5 degrees of full flexion and extension, while supination, pronation and the carrying angle were normal (fig. 2).

Case 2. A boy, aged 7, was admitted to a hospital with an injured elbow. A reduction had previously



CASE 2. FIGURE 3.
Original fracture.

been attempted by a local physician but without the help of an x-ray examination. The boy, on his arrival at the hospital 3 hours later, showed an acutely swollen elbow. The hand was warm and the color fair, but no pulse was discernible at the wrist. X-rays showed a typical supracondylar fracture of the humerus with the usual displacement (fig. 3). Two

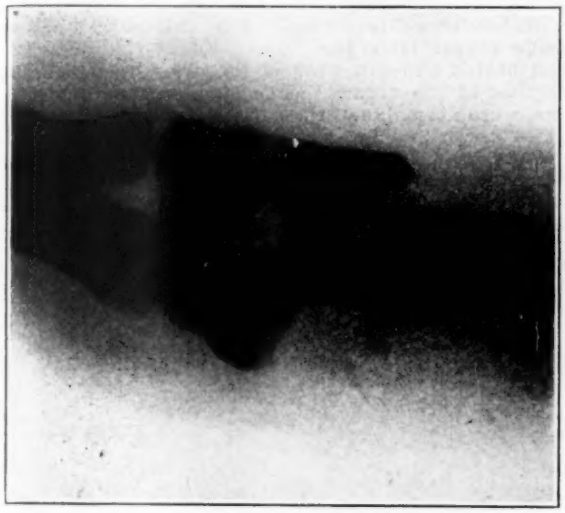


CASE 2. FIGURE 4.
End result 1 year later.

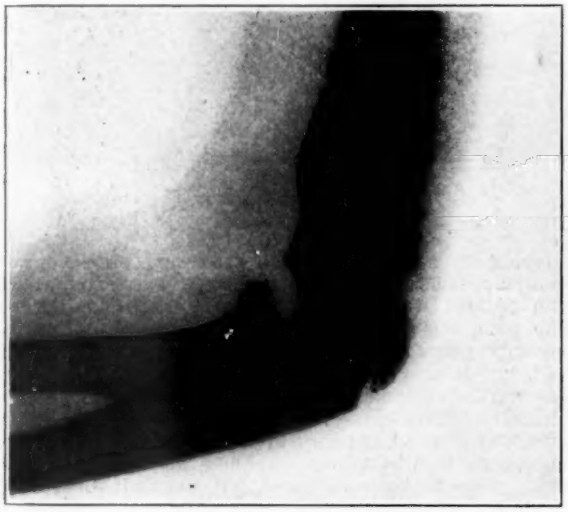
unsuccessful attempts to reduce the fracture were made at the hospital. He was seen by the writer 48 hours after the injury, at which time he had a tremendously swollen elbow. There was no radial pulse, the hand was a little cold, and there was cyanosis of the fingertips. The patient was given ether anesthesia, and an incision was made which split

the triceps tendon. The fracture site was exposed, and a large amount of clotted blood was removed. The proximal fragment was delivered, and the anterior aspect of the joint cavity was explored. The fragments were then apposed, after which the elbow could be placed in acute flexion, although it was finally put up halfway between acute flexion and a right angle. When the arm was inspected a few hours after the operation, the hand was warm but there was still no pulse at the wrist, although cyanosis was absent. Careful watch of the hand was kept for a period of 48 hours, after which time the pulse reappeared. An examination 1 year later showed that extension and flexion were within 5 degrees of the normal range, while the carrying angle, supination and pronation were good (fig. 4).

Case 3. A boy, aged 8, was injured in falling from a bicycle. He was taken immediately to a hospital,



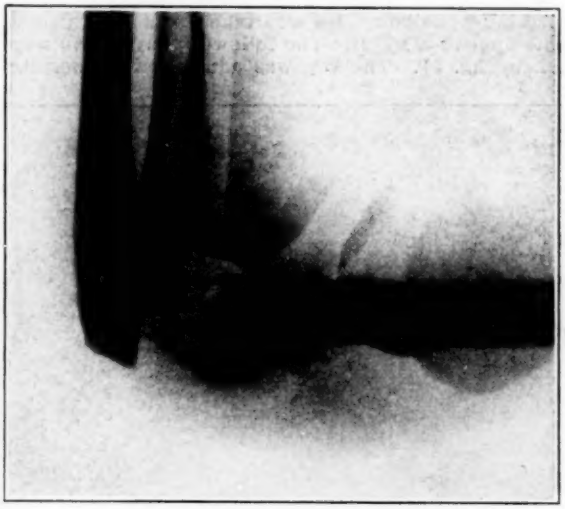
CASE 3. FIGURE 5.
Original fracture.



CASE 3. FIGURE 6.
After operation.

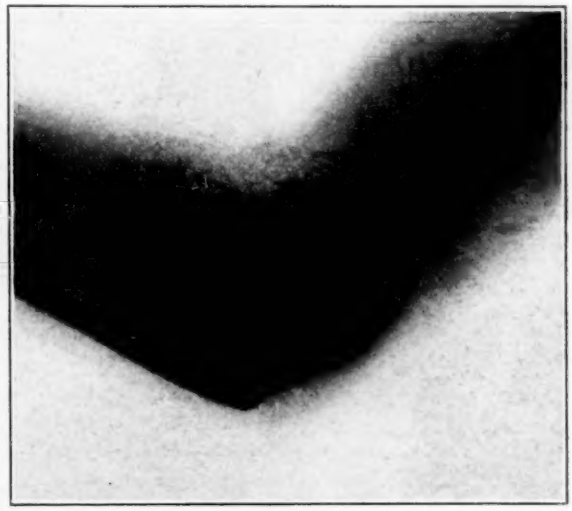
where x-rays were taken (fig. 5), and a reduction of the fracture was attempted. The patient was seen by the writer the following day, at which time he showed cyanosis of the arm and coldness and swelling of the hand. The pulse was barely palpable. The arm was placed in full extension. Twenty-four hours later the pulse was good, and the circulation had improved. An open reduction of the fracture was done through a posterior incision. The elbow was brought up in acute flexion, and the pulse at the wrist was good. A final check-up (fig. 6), 1 year later, showed all motions of the elbow to be normal.

Case 4. A boy, aged 5½, fell 6 feet, landing on his outstretched arm. He was taken to a hospital immediately. At the time of admission his left arm was



CASE 4. FIGURE 7.
Original injury.

held in full extension, with a prominent swelling in the antecubital fossa, the pulse being palpable at the wrist. An x-ray check-up revealed a fracture (fig. 7), reduction of which was attempted under

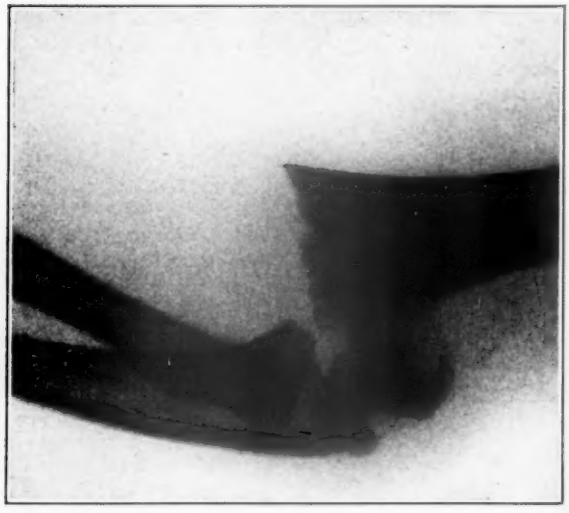


CASE 4. FIGURE 8.
Final check-up 1 year later.

a general anesthetic. Following this, the pulse could not be felt at the wrist. The arm was then brought down, and ice bags were applied to it. Three days later the arm was put out in extension and was watched for about a week on account of the swelling. Eighteen days after admission, and following two

manipulations, the writer was asked to see the patient. The position of the fracture as shown in the original x-ray had not been changed, and it was decided to do an open operation. A split triceps tendon exposure was made and, in spite of a very small amount of callus, the fracture was easily reduced and the arm put up in acute flexion. The radial pulse was good. The final check-up, 1 year later, showed only slight limitation of motion (fig. 8); the elbow went to within about 10 degrees of full extension, while supination and pronation and the carrying angle were good.

Case 5. A boy, aged 9, fell from a height of 4 feet, striking his elbow. He was taken to a local doctor, who applied a splint. The following day x-rays were taken (fig. 9). The boy was admitted to a hospital



CASE 5. FIGURE 9.
Original fracture.

CASE 5. FIGURE 10.
Final check-up 1 year later.

with a very marked swelling of the elbow, with blebs and blisters on the skin locally. The temperature was 102° F., and there was no pulse at the wrist. The blebs and blisters on being opened discharged a semipurulent material. The pulse at the wrist was watched very carefully until its return to normal at the end of 48 hours. The blebs and blisters were dressed for a period of 22 days. By this time the swelling had subsided, and the skin looked perfectly clean posteriorly. A split triceps tendon incision was then made. The capsule was found to be markedly distended with blood clot, and the callus formation was fairly well advanced. The proximal fragment was found to be displaced anteriorly, and it was with considerable difficulty that the callus was freed and the fragments apposed. At this stage the elbow could be put in acute flexion without any difficulty, although it was discovered that the radial pulse was absent. About 8 hours later, as the pulse had not returned, the wound was opened again, and it was found that anteriorly there was still some pressure from the callus, which had not been removed. The callus was excised, and the elbow was again put into acute flexion. The following morning the pulse was perceptible but very faint, but by the fourth day it had returned to normal. The final check-up was made 1 year later (fig. 10). At that time the boy had flexion and extension of the elbow to within 10 degrees of the normal, while supination and pronation were normal, and the carrying angle was good.

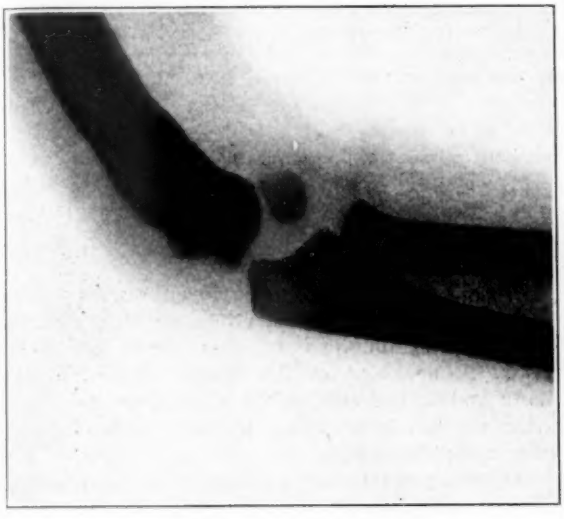
Case 6. A boy, aged 4, slipped and fell, striking his right elbow. His mother observed a deformity and took the child immediately to a doctor, who took x-rays of the elbow (fig. 11). The child was then

CASE 6. FIGURE 11.
Original fracture.

referred to a hospital. The reduction of a supracondylar fracture of the humerus was attempted. The patient was seen by the writer 2 days later. The arm was considerably swollen, and the pulse was not palpable at the wrist. The hand was not cold, but it was a little dusky in color. An ice bag was applied to the swollen elbow with the arm in full extension. The pulse returned in 24 hours, and the condition of the hand improved. Eighteen days after admission, a split triceps incision was made in the arm. Some callus was found, but the proximal fragment was delivered without any difficulty. The arm went readily into acute flexion, and the radial pulse was of good quality. The end result at the time of the patient's discharge, 4 months later, was a good carrying angle and no loss of supination or pronation, while the elbow went to within 5 degrees of full extension and flexion (fig. 12).

There is a great deal of material in these cases that is interesting to analyze. The first case, for example, strongly suggests that it would be advisable to obtain a history of recent illnesses before attempting operative procedure on joint cases. It is the writer's firm belief that the infection in this case was not a complication of the operative technic, but rather a residual from the previous attack of scarlet fever. It is needless to say that this patient gave the writer an extremely uneasy 3 or 4 weeks, and it is to be regretted that no culture of the wound was taken.

A common factor in the histories was the repeated attempt at closed reduction, with force applied improperly, both quantitatively and



CASE 6. FIGURE 12.
End result 4 months later.

mechanically. There was a great deal of traction and attempted manipulation of the fragments with the elbow in full extension. Gentleness in handling traumatized tissues about fracture lines cannot be too strongly emphasized. When one stops to consider the delicate structures contained in the antecubital space, the caution should be applied to the greatest possible degree. Obviously, traction should always be applied with the elbow at a right angle. The force should be gentle but firm and should be maintained in the line of the long axis of the humerus. The force should be sufficiently prolonged so that one is reasonably sure that the proximal fragment will ride by and become apposed to the distal fragment when the elbow is brought into flexion. This may be accomplished in many ways, either by the operator holding the elbow between his fingers with the assistant exerting countertraction on the humerus, or by overhead traction with a Kirschner wire through the ulna. The point to be emphasized is that the elbow must be at a right angle.

The second common factor in these cases was the absence of the radial pulse, with the additional factors of a warm hand and the absence of skin anesthesia. Perhaps it is not overestimating the gravity of supracondylar fractures to say that all of them are potentially Volkmann's contractures in unskilled hands. The elbow is endowed with a very efficient collateral circulation that withstands, up to a certain point, constriction of the brachial artery, but it does not have a collateral nerve supply. It is the general belief that a true Volkmann's contracture is the result of sustained pressure on the blood supply to the forearm and hand, although, unquestionably, in extreme cases, pressure on a nerve trunk plays an important role. With this in mind it is apparent that a patient whose radial pulse is absent demands constant observation by a trained surgeon until the latter has thoroughly convinced himself that the collateral circulation has been established. The longest period before the radial pulse returned after operative procedure was 3 days, and at no time was the circulation of the hand impaired to the extent that it produced a difference in temperature. The intra-articular pressure in the average case is the result of a hemorrhage from the site of the fracture, plus the fact the displaced proximal fragment presses on the anterior portion of the capsule of the elbow joint and compresses the brachial artery between the capsule and the antecubital fascia. Because of the anatomic distribution of the fascia it does not have too much elasticity. As a result, the vessels and the median nerve are really compressed between two nonresisting substances. It is not known in how many cases the anterior portion of the capsule is torn. Such a tear permits the intra-articular hemorrhage to pass into the antecubital space. This condition was not found to be present in any of the cases in this group.

A third common factor that was concerned with treatment was that a posterior split triceps tendon incision was made in all but 2 of these cases. In the writer's opinion, the advantages of this incision are the ease of approach, the absence of trauma to the tissues and the simplicity of reduction of the fracture. In each case, the joint was found to be full of blood clot, but a small skid was placed under the proximal fragment which could then be tipped back onto the distal fragment without difficulty. Another advantage of this procedure is that the joint can be inspected and, also, the antecubital fossa palpated so that any distention of that region can be noted.

The fourth common factor was that there were no resulting "frozen elbows" in this series. There were, at most, only about 5 or 10 degrees of limitation in flexion and extension, with no limitation in supination and pronation in any case.

A fifth common factor was the treatment by

physiotherapy. At no time in the postfracture treatment was force applied to increase the motion at the joint. The treatment was confined to baking and massage, with active weight suspension or overhead suspension exercises performed by the patient and associated with game activities outside.

It is the writer's belief that all attempts at closed reduction in supracondylar fractures of the humerus should be confined to one manipulation in skilled hands, after which, if this manipulation is not successful, a posterior incision should be resorted to. It is also his belief that a displaced proximal fragment as well as the intra-articular hemorrhage plays an extremely important part in presenting a firm, resistant back wall, which, when compressed against the antecubital fascia, may cause harmful nerve and vascular pressure. It is a well-known fact that a Volkmann's contracture may occur irrespective of the presence of a fracture in the bones of the arm. In such cases, however, we find a definite injury somewhere along the course of the artery. Obviously, in the cases in which there is not only absence of the radial pulse but also skin anesthesia and coldness of the hand, there can be no question in the mind of any experienced surgeon but that the fascia should be incised anteriorly, particularly if the posterior incision has not relieved the condition. It is the writer's belief, however, that a posterior incision, followed by a reduction of the fracture and the removal of the blood clot from the joint will, in many cases, clear up a group of fractures that frequently result in tragedy under other circumstances.

NUTRITION AND PUBLIC HEALTH

"More money for food, and more effective application of present knowledge of food and nutrition to food selection problems, are necessary if higher levels of nutrition are to be more generally attained by low income city families," Dr. Hazel K. Stiebeling, senior food economist of the Bureau of Home Economics, U. S. Department of Agriculture, said recently in an address at the round table conference on nutrition and public health, held at the New York Academy of Medicine under the auspices of the Milbank Memorial Fund.

Summing up the results of a recent Bureau study on the diets of wage-earners and low-salaried clerical workers in North Atlantic, Southern and Pacific Coast cities, Dr. Stiebeling said:

"Major differences in the amount and kind of food purchased by city families can be attributed to differences in the level of expense for food. With increasing expenditures for food, there is a marked increase in the total amount of all food purchased. The increases are much more marked in eggs, meats, milk, butter and the succulent vegetables and fruits than in grain products, potatoes, dried legumes and fats other than butter. This unequal distribution of the increases in food purchases with increasing expenditure for food results in more food for higher income groups, and in general, food richer in high-quality proteins, in minerals, and in vitamins.

"High expense for food is not necessarily a guarantee of adequate diets. The study showed that at spending levels between $1.50 and $4.50 per week per food-cost unit, some families managed to get 'grade A' diets, some had 'grade B' diets, and others had 'grade C' diets.

"As more money is spent for food, families tend to buy more of the milk, butter, eggs, succulent vegetables, fruits and other foods which are good sources of the calcium or certain vitamins in which the most frequent shortages were found among the diets studied.

"Of the wage-earner families studied, about 10 per cent of those living in the North and far West, 38 per cent of the Southern white families and 68 per cent of the Negro families spent for food less than $2.00 weekly per food-cost unit. About 20 per cent of the families of wage-earners in the North and far West, 6 per cent of the Southern white families and less than 3 per cent of the Negro families spent as much as $4.00 weekly per food-cost unit.

"About one fifth of the employed workers' families in the North Atlantic cities, an eighth of the Pacific Coast families in this same population group, 40 per cent of the white workers in the South and 70 per cent of the colored workers were living on 'grade C' diets, if the families studied were a typical sample.

"At 1935 price levels, apparently no families had better than 'grade C' diets unless they were spending as much as $1.25 in the North Atlantic region, or $1.75 in the South, weekly per food-cost unit. Some families seemed to require as much as $4.00 in the North and West and $3.50 in the South to secure fair or adequate diets. Average diets costing about $3.00 weekly per food-cost unit in the North and West, and about 10 per cent less in the South, were at 'grade A' level, but less than half the families at this spending level had 'grade A' diets.

"In the Pacific region, a higher percentage of families at each expenditure level seemed to get good diets, and a lower percentage had poor diets, than in other regions. At every level of food expense, Pacific families bought more vegetables and fruits than did families in the North Atlantic region, about the same amount of eggs and milk, less meats, and a smaller share of their grain products in highly processed forms."

MATERNAL MORTALITY

"Lying-in is neither a disease nor an accident, and any fatality attending it is not to be counted as so much per cent of inevitable loss. On the contrary, a death in child-bed is almost a subject for an inquest. It is nothing short of a calamity which it is right that we should know all about, to avoid it in the future."—*Florence Nightingale.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23191

PRESENTATION OF CASE

A 23 year old American housewife was admitted complaining of pain in the chest.

About 1 year before entry the patient developed dull, constant pain in the left chest anteriorly and at the left costal margin. The pain gradually increased in severity and began to radiate to the left shoulder and down the left arm. As the discomfort increased, the radiation became more widespread, and she began to suffer from transient knifelike pains in the region of the left costal margin, radiating to the back. About 2 months after the onset she developed what was termed pneumonia; the illness was associated with a chill, increased chest pain, cough and rusty sputum. At that time she remained in bed for 3 weeks and, a short time later, was again confined to bed because of a left pleural effusion. During the succeeding 3 months her chest was tapped on several occasions, and from 1 to 2 liters of fluid was removed each time. About 6 months prior to admission, following slight exertion, she suddenly experienced knifelike pain in the left chest, which was accompanied by extreme dyspnea and fever. She was informed that she had a collapsed lung. Thoracentesis produced relief. The symptoms continued with varying intensity, and during the month before coming to the hospital she developed anorexia, severe nausea and vomiting. There was frequent gaseous eructation, and the patient complained that her stomach filled rapidly, even after 4 or 5 teaspoonfuls of food. There was no cough at the time of entry. Her weight had diminished from 135 to 116 pounds during the 2 years preceding entry.

Six years before coming to the hospital the patient had a curettage performed because of profuse metrorrhagia and menorrhagia. This produced no relief, and a partial oophorectomy for ovarian cysts was done. There was a slight abatement of her symptoms thereafter, but 2 years later another laparotomy was performed, and a bilateral salpingo-oophorectomy was done. There were said to be many small ovarian cysts and adhesions in the region of the previously removed appendix. Three months before entry a small tumor was removed from the subcutaneous tissue of the right shoulder. The sections from this tumor showed fibroma.

Physical examination showed a moderately emaciated, pallid young woman, who complained of considerable nausea. The skin was well tanned from previous sun irradiation. The neck veins were not engorged. The chest was asthenic in configuration, and expansion was limited on the left side, although slight fullness of the left lower chest was noted. Posteriorly there was percussion flatness from the angle of the left scapula to the base, and anteriorly similar flatness from the fourth left rib downward. Breath sounds in this region were absent, and tactile fremitus and vocal resonance were diminished to absent. The heart was not remarkable, and the blood pressure was 100/80 in both arms. The remainder of the examination, including a pelvic examination, was negative.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 5,500,000, with a hemoglobin of 80 per cent. The white cell count was 15,900, 81 per cent polymorphonuclears. Repeated examinations of the sputum showed occult and macroscopic blood, but no specific microorganisms were found. A blood Hinton test was negative. The sedimentation rate was 4 millimeters per hour.

Review of the previous x-ray films showed left pleural effusion with two or three sharply defined, rounded nodules in the left lung and one in the right. At the time of entry a film showed increased fluid on the left side with displacement of the heart to the right. There was questionable cardiac enlargement. The gas bubble of the stomach was displaced downward, and there was enlargement of what appeared to be the spleen. X-rays taken with Bucky diaphragm showed rather large, irregular areas of calcification in the extreme posterior aspect of the left lung; the other masses did not show calcification. There was a rounded area of calcification in the soft tissues of the left inguinal or gluteal region, but there were no other similar areas in the left lower extremity, and the bones were normal. Examination of the esophagus exhibited no definite evidence of obstruction, although it was displaced slightly to the right. The fundus of the stomach and the shadow of the spleen were low in position.

Shortly after entry a left thoracentesis produced 600 cubic centimeters of dark yellowish, clear fluid, the specific gravity of which was 1.015. The cell count showed 40 white blood cells per cubic millimeter, of which 50 per cent were lymphocytes, 25 polymorphonuclears and

25 monocytes. There were also 40 red blood cells. A culture showed no growth. The patient was given a course of x-ray treatments to the thorax, and her temperature rose gradually until it leveled between 102 and 104°F. during the third and fourth weeks in the hospital. X-ray examination showed dulness at the right base but no change in the left side of the chest. The heart shadow was definitely enlarged to the right, and no cardiac pulsations were seen. A right thoracentesis produced 950 cubic centimeters of grossly bloody fluid. The patient had previously developed a cough productive of blood-stained sputum, which on occasion was purulent and odorless in character. Postural drainage was instituted, and the purulent sputum disappeared. At the end of a month in the hospital another chest film showed a gas bubble in the right side of the chest, just above the diaphragm. This contained a shifting fluid level, and its margins were markedly irregular and rounded. There was no definite evidence of air in the pleural cavity. Several smaller, less distinct, air- and fluid-filled cavities were seen above the large cavity. There was a small amount of air in the left pleural cavity, but there was no increase in fluid on either side. The patient gradually developed edema of the lower extremities and evidence of fluid within the abdomen. On several occasions she had attacks of severe dyspnea and collapse, during which apparently only the administration of stimulants and oxygen preserved her life. During the second month in the hospital, her temperature fluctuated between 98 and 102°F. and then gradually reached a normal level. The white cell count, which had risen to 21,000, gradually diminished to 11,000. A left chest tap was performed, and 500 cubic centimeters of sherry wine-colored fluid, in which there were 8,000 red blood cells and 70 white blood cells, was withdrawn. An x-ray taken on the fifty-first hospital day showed that the cavity on the right side had decreased in size and was half filled with fluid. The left lung was unchanged. A week later an area of dulness appeared in the midportion of the right lung, and the dulness on the left side had increased. Two weeks later the right-sided dulness had for the most part subsided, and fluid on the left side had diminished. She was discharged, without cough, fever or cyanosis, on the seventy-fourth hospital day.

Following her discharge the patient led a bed and chair existence and ate very little food. About 3 weeks after discharge, she began to fail rapidly and developed marked edema of the legs, swelling of the abdomen and pronounced anorexia. Thirty-eight days after leaving the hospital she had a sudden attack of extreme dyspnea and lost consciousness for a short while. A physician was called and found her markedly dyspneic and cyanotic. There was a small amount of fluid at the left base, and the heart dulness was increased. The heart sounds were rapid but of good quality. She died several hours after the onset of the acute episode, 1 year and 5 months after the onset of her chest symptoms.

Differential Diagnosis

Dr. Soma Weiss:* May I ask one question? Had this woman had any pregnancy in the past?

Dr. Francis T. Hunter: Definitely, no.

Dr. Weiss: That makes a difference.

In order that we may understand the clinical course of this patient, we have to establish two points: first, the fundamental nature of the disease and, secondly, the character of the functional and morphologic changes in the body which were responsible for the multiple symptoms and signs exhibited by the patient.

You will agree with me, I hope, if I say at once, without giving explanation, that the fundamental nature of this disease was neoplastic, with an overwhelming localization in the thorax. The first difficulty arises when we attempt to ascertain whether the neoplastic disease in the thorax is primary or metastatic. I should say, statistically, that the symptoms presented by this patient would favor the diagnosis of primary neoplastic disease of the lungs because hemoptysis, purulent sputum and cavitation occur relatively frequently in primary carcinoma of the lung, while they are quite rare in metastatic disease. Against the concept that this is a primary neoplastic disease of the lungs, however, there are two kinds of evidence: first, the patient had certain difficulties with the functions of the genital organs, and certain peculiar cystic tumors were found in the neighborhood of the appendix during laparotomy and, secondly,—and this is the most weighty evidence,—the first x-ray taken revealed three sharply demarcated, definite, round shadows on the left side of the chest and one round shadow on the right. I shall place greater weight on this finding than on the statistical evidence. Therefore, I believe that the neoplastic disease in this patient is not primarily intrathoracic but metastatic in the chest. We have very little evidence as to the nature of the primary tumor, and, therefore, I should like to drop this problem for a moment and return to the analysis of the symptoms and signs exhibited.

The patient had particular difficulty with thoracic pain. She had several kinds of pain, localized and radiating to the left and to the back. That sort of pain is more apt to develop with primary than with secondary neoplasms of the lungs. We know, on the other hand, that metastases, particularly those that grow rapidly and invade structures freely, can give pain, although in the majority of instances neoplastic

*Associate professor of medicine, Harvard Medical School. Assistant director, Thorndike Memorial Laboratory, Boston City Hospital.

disease of the thorax characteristically causes very few symptoms and signs. Nausea and vomiting in this patient do not help us because we do not know whether these symptoms were due directly to pressure or were indirect factors. Nausea and vomiting can be produced by a great many mechanical and physiologic factors in all parts of the body. In this patient there was evidence that the stomach was displaced, as was also the spleen, and this alone is sufficient to explain the nausea and vomiting. There is an interesting and somewhat unusual finding in the thorax as described, and that is the calcification which was found on the second examination and which was not present at the time of the first examination. Calcification of neoplastic tumors occurs, particularly in the slowly growing and hemorrhagic tumors. At times, calcification forms a peripheral ring if the tumor is cystic; I have seen such calcification also with echinococcus cysts. There are also other types of tumors that may calcify, such as certain sarcomas.

As far as the hemorrhage, the purulent sputum and the fever are concerned, they all belong together; and, in spite of the fact that these changes are apt to occur in primary neoplastic disease, as I said before, they can occur with metastases provided that the latter invade and perforate the bronchus and begin to bleed within the bronchial canal, when there is a chance for secondary necrosis. Such changes can lead to all the symptoms mentioned here. The observation of, first, clear fluid and, later, bloody fluid does not require further explanation.

There is, however, a group of symptoms which are somewhat unusual in neoplastic disease of the thorax, and I refer to dyspnea, collapse, loss of consciousness, cyanosis and manifestations of dilatation of the right side of the cardiac shadow, which did not pulsate, and some displacement of the esophagus to the right. In other words, this patient had difficulty with her circulation, and that raises a very important and interesting question. *What are the metastatic or neoplastic cardiac complications in general, and which of these can produce cardiovascular and circulatory difficulties?*

Neoplasms producing circulatory difficulties are numerous. They can be localized within the heart with the formation of myocardial metastases. By direct invasion a tumor can involve the pericardium and produce pericarditis with or without fluid. The endocardium may show implantations of the neoplasm and formation of a thrombus, which may produce obstruction, sometimes on the right side of the heart. Rarely, a tumor, particularly from the mediastinum, may directly invade all three layers of the heart and produce the changes as described. Finally, coronary occlusion can occur in the form of neoplastic cells. There is another group of complications where the inflow is disturbed in either the superior or the inferior vena cava. The third group of complications is where the outflow from the right side is disturbed by changes within the lungs. This type of change consists of the so-called carcinomatous lymphangitis, where all the lymphatic vessels around the pulmonary arteries become involved; the vessels become choked, the small arteries may become occluded by neoplastic cells, and secondary thrombotic manifestations following a proliferative pulmonary endarteritis are not uncommon. Finally, emboli may be discharged from an intracardiac neoplastic thrombosis.

The circulatory symptoms exhibited by this patient cannot, in my opinion, be explained either by a simple intramyocardial metastasis or by a coronary occlusion, because we know from experience that the majority of these complications are usually silent and, if they produce symptoms, they usually cause arrhythmia. It seems to me that the most plausible explanation for the circulatory symptoms is either that there was some difficulty with the inflow to the right side of the heart or that there was some difficulty in the outflow, such as might be caused by a pulmonary embolus. At least I believe that the x-ray picture is not compatible with neoplastic lymphangitis, which is so frequently associated with syncope, attacks of dyspnea and similar symptoms. Therefore, on the whole, I favor a neoplastic thrombus on the right side of the heart from which possibly metastatic emboli have dislodged, and I believe that we are dealing with a *metastatic neoplastic tumor involving both lungs, the pleura and also the heart.*

The question finally is, if we are right in thinking that this is not a primary intrathoracic tumor, *Where is the primary tumor and what is the nature of the neoplasm?* The possibilities are, in my opinion, tumor of the stomach, the kidney or the genital organs, or bone sarcoma. For primary renal neoplasm we have no evidence whatsoever. Therefore, I cannot make such a diagnosis. For a neoplasm of the stomach, we have little evidence of symptoms, and we can account safely for the symptoms we have by neighboring pressure manifestations. That leaves two conditions for differential diagnosis: (1) malignant neoplasm originating in the genital organs and (2) primary bone cell sarcoma. Among the tumors of the genital organs of the female, choriocarcinoma can explain practically all the symptoms because in about 70 per cent of the instances a choriocarcinoma has manifestations in the lung, and metastases from choriocarcinoma are notoriously hemorrhagic. They are apt to form embolic manifestations in the lung in the form of villi, and one can get cavities and even invasion of the heart. But if it is true that this patient had no pregnancy, we can rule out such a diagnosis fairly safely. There

are reported here and there cases of choriocarcinoma in young, single, female patients, but whether such cases are authentic is highly questionable. These choriocarcinomas occur in males from teratomatous origin. Such reasoning leaves a final diagnosis of primary bone sarcoma. Bone sarcoma, almost as frequently as choriocarcinoma, forms diffuse metastases in the lung. The metastasis may start with a round shadow, grow extensively, break into the bronchus, form pulmonary cavities and may even invade all layers of the heart either directly from the mediastinum or indirectly through the circulation. Furthermore, the few calcified areas in the soft tissue may also be explained by a sarcoma, while they would be difficult to explain by a choriocarcinoma. If this was a sarcoma, what were the changes described in the pelvis? I do not know. They may have been an irrelevant finding or the patient may have had an unusual form of endometriosis. In conclusion, I believe that we are dealing with a *primary bone sarcoma with diffuse metastases in the thorax, involving the lung, the pleura and probably the right side of the heart, from which perhaps embolic manifestations may have developed, finally leading to right-sided failure.*

DR. AUBREY O. HAMPTON: These are the films brought in by the patient. She had had a chest tap and had air and fluid on the left side through which we could see the nodules described. This one was calcified at that time, though it is not in the record. This was in October, and the following January the nodule in the right chest had remained about the same size. At our first examination the main difference appeared to be the right border of the heart, which is either displaced or enlarged, and the gas bubble in the fundus of the stomach is flat. This is a film taken about 1 month later, following the episode of fever, bloody sputum, postural drainage and so forth, and here we see the cavities, apparently in the right lung. As I understand it, there was no chest tap on the right side before this film was taken.

DR. HUNTER: Yes, she had had a chest tap on that side.

DR. HAMPTON: At any rate, we took these films to prove that the lesion was in the lung. I think it does. I never saw a pleura so irregular as that, and I never saw a fluid level stop so abruptly. We were quite sure she had an abscess in the lung, which was not in the area of tumor. Fifteen days later the abscess was reduced in size, and the left side of the chest improved, I think from chest tap. The mass in the right side remained about the same size during these 2 months.

I do not have the film that was taken immediately after one of the attacks of acute cyanosis, but this one does show an increase in dulness. This one was taken some time later and shows a marked increase in dulness on both sides. Here you can see the areas of calcification. One mass was 4 centimeters in diameter with other similar ones above it; they were against the chest wall but apparently not in the pleura. Here is the mass, and there it has the shape of one of those nodules and is composed of numerous small areas of calcification, with linear and irregular areas extending out from it in all directions. Here is the stomach, which is displaced downward the distance of at least three ribs. This is the shadow of the spleen. The bones were searched for metastases, but we found none. We did find this small area of calcification in the inguinal area which is quite similar to this one in the chest. We then proceeded to examine the extremities carefully, thinking of course that an osteogenic sarcoma was present in the lower extremity, but it was not. The bones were perfectly normal.

DR. WEISS: I must say that the picture looks a little different from the way I imagined it from the description, and in view of the x-ray pictures I would like to raise in the differential diagnosis the possibility of some parasitic infection, such as echinococcus cyst. Was she an American housewife?

DR. TRACY B. MALLORY: Yes.

DR. WEISS: I have never seen such diffuse echinococcus cysts in an American. So, on that basis, I shall not raise the possibility. I maintain my former diagnosis.

DR. HAMPTON: I forgot to mention the heart. I think it was perfectly obvious that the heart was enlarged, and here we had the impression between these two films that it changed in size. There were no cardiac pulsations at the time she was first seen.

DR. HUNTER: This was my patient, and I would like to make a few additional comments about the history. However, I do not think they would make one change one's mind.

First of all, she was tapped nine times before she came in. Secondly, from hearsay, one doctor said that she had had an adenocarcinoma of the ovary. Thirdly, I never felt the spleen. Fourthly, guinea-pig inoculation of the pleural fluid was done outside and was reported as negative. After her discharge from the hospital I saw her at home. There were no signs of fluid. Her cardiac dulness was exactly the same. The sounds were fairly good. The neck vessels were not distended. I mentioned to the family that she might have metastasis in the heart because she showed cardiac failure without the ordinary explanation for it. Finally, to say that she had blood-stained sputum is incorrect. She was spitting up as though she had a lung hemorrhage, and that fact led me to make a tentative diagnosis of hemangiosarcoma of the lung with calcification. However, in view of the information I received later about the pelvis we made

a final diagnosis on the death certificate of adenocarcinoma of the ovary.

DR. MALLORY: Are there any other suggestions?

DR. HAMPTON: I would like to have a discussion of the incidence of calcified metastatic nodules in the lung, other than osteogenic sarcoma. I do not remember having seen metastatic malignancy to the lung produce calcification unless it was primary in the bone.

DR. WEISS: Renal carcinomas are described, and I have seen them produce, not calcification of this sort, but delicate ring-like calcification.

DR. MALLORY: It is very common in a wide variety of primary tumors, but I think I would agree with Dr. Hampton that it does not often occur in metastases, except in bone tumors.

CLINICAL DIAGNOSIS

Carcinoma of the ovary, with metastases to the lung and generalized carcinomatosis?

DR. SOMA WEISS'S DIAGNOSES

Sarcoma—primary in bone.
Metastases in the lungs, the pleura and the right side of the heart, with right-sided cardiac failure.
? Neoplastic pulmonary embolus.

ANATOMIC DIAGNOSES

Fibrosarcoma involving lungs, mediastinum, pericardium, heart and jejunum—? von Recklinghausen's disease.
Compression of esophagus, aorta and superior vena cava.
Hydrothorax, left.
Hydropericardium.
Operative scars: excision of fibroma of right arm; bilateral salpingo-oophorectomy; appendectomy.
Chronic passive congestion, liver and spleen.
Edema of the lower extremities.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The autopsy on this case answers some questions, and leaves some issues still unsettled. At any rate, we found widespread tumor. The major lesion was in the left lower lobe of the lung. That was calcified and was directly continuous with a tumor mass which infiltrated the entire parietal pleura of the left pleural cavity. It went directly through the pericardium, producing a complete replacement of the pericardium with tumor. The pericardium contained approximately a liter of bloody fluid. The heart was very small and was covered with epicardial metastases. There was one metastasis, measuring 1 centimeter in diameter, subendocardially in the pulmonary conus. The tumor had invaded the mediastinum very diffusely, and a large tumor mass was found above the heart enclosing the pulmonary artery and, to some extent, the aorta, producing rather marked narrowing of the superior cava; the inferior cava was free. Just above the diaphragm the tumor again encircled the aorta and, at this point, very markedly reduced its caliber. An obviously metastatic tumor nodule was found in the right lung. We did not find any sign of an abscess. The pleural cavity on that side was totally obliterated, and the lung, except for metastases, was entirely negative. So I am inclined to think that that original bubble of air was in the pleura and not in the lung.

DR. HUNTER: She was tapped twice on the left and once on the right.

DR. HAMPTON: I cannot agree with Dr. Mallory's statement.

DR. MALLORY: If it were in the lung, it had completely healed and had left no scar.

DR. HAMPTON: She absorbed air on the left side very quickly. She still had a gas bubble—over a period of 1 month.

DR. MALLORY: I will have to leave that open. The liver showed a very marked degree of passive congestion but no metastases. None were found in any of the peritoneal organs. There were ascites and peripheral edema, unquestionably the result of circulatory failure and not due to carcinomatous involvement of the serous surfaces. All these tumors looked very much alike histologically and were essentially similar to the tumor which was removed from the left arm. In most areas they looked like perfectly benign fibromas. In a few areas they were more rapidly growing. The only spot, however, where tumor was growing rapidly enough to be obviously sarcomatous was a small ulcer in the jejunum, which was thought to be a benign ulcer on gross examination, but which microscopically proved to be a metastatic tumor. It was small, and could not have been the primary source. So we are left with the question of where this tumor was primary. There are three possibilities. It is perfectly consistent with metastasis from a primary fibrosarcoma of the ovary; we have never been able to get the original slides of her ovarian tumor, but the description would seem to rule out that possibility, since ovarian fibrosarcomas are solid, not cystic, tumors. It might be a single fibrosarcoma with multiple metastases, and in that case the most probable source is the pericardium, with direct extension both ways into the lung, the mediastinum and across the pericardium into the heart. That would not particularly well explain the apparently benign but very similar looking tumor in the arm, in the region of the deltoid muscle. Our third possibility is that we have multiple foci of origin of the tumor. In other words, we are dealing with a von Recklinghausen's tumor. The tumors are not characteristic of neurofibroma microscopically, but von Recklinghausen's disease

varies so much in histology that I am not confident that I can always recognize it in sections.

I have two pictures that I would like to show. The first (fig. 1) is the anterior aspect of the thoracic organs. The main tumor mass is behind the heart and, therefore, does not show,

FIG. 1. Case 23191.

but several of the tumor masses in the epicardium are evident, and a pedunculated tumor mass is apparent between the right border of the heart and the lung. The tumor surrounds the great vessels at the base of the heart, and an incision has been made through it to open the pulmonary artery. The second picture (fig. 2) is the posterior aspect of the thoracic organs. The probe lies in the aorta, which has been completely encircled by the tumor. The right lung has been incised and a portion of it folded back to show the complete involvement of the lower lobe.

I am sure Dr. Weiss was correct in assuming that the circulatory mechanism was impaired. Which of these various points of involvement was actually responsible for the symptoms, I do not think we can say. From the autopsy the pericardium looked like the most severe lesion, but I do not think it fits the clinical symptoms very well.

DR. WEISS: If the observation that the venous pressure in the neck was not elevated is correct, that is very strongly against any appreciable degree of pericardial compression which could produce syncope. Certainly the myocardial involvement is very probably responsible. I think the endocardial involvement alone, or combined with the embolic metastasis, is the most plausible source of circulatory failure.

FIG. 2. Case 23191.

DR. MALLORY: We did not find any emboli in the pulmonary vessels. The branch to the left lower lobe was completely occluded from external pressure, but the other branches were all free.

A PHYSICIAN: You mentioned something about the pulmonary artery?

DR. MALLORY: The main pulmonary artery was completely surrounded by tumor but did not seem to be markedly narrowed by it.

CASE 23192

PRESENTATION OF CASE

A 50 year old native factory worker was admitted complaining of pain in the right flank.

The patient had always been well until 2½ years before entry, when he had an acute attack of severe pain in the chest, which was diagnosed by a physician as coronary thrombosis. He remained in bed for 6 weeks and then had an attack of phlebitis which confined him to bed for another 3 weeks. About 2 years before coming to the hospital, he began to experience

a sensation of abdominal distention associated with nausea whenever he was particularly tired or nervous. These attacks caused vomiting and were also relieved by it. The vomitus was liquid in character and contained bile. The attacks occurred about fifteen to twenty times weekly, during or directly after meals, and were accompanied by frequent eructations. At this time he noted the gradual onset of intermittent pain in the flank beneath the right costal margin. The pain varied in intensity and was occasionally knifelike in character and awakened him from sleep. There was no evident relation between the pain and the vomiting. There was tenderness in the right upper quadrant and over the second lumbar vertebra. The discomfort continued, and a year and a half before coming to the hospital he was compelled to discontinue work because of weakness. At that time he eliminated meats and fried foods from his diet and under this regime felt somewhat improved and returned to work. After 4 months he was again forced to discontinue work and to rest for several months. Periods of respite from discomfort and of recurrence of pain occurred until entry. Rest always relieved his symptoms. Four months prior to admission he had an acute attack of pain in the right lower quadrant, and a gangrenous appendix was removed. Repeated stool examinations during the last few months showed no blood, and a gastrointestinal series, a barium enema and gall bladder tests were said to be negative. His weight decreased from 156 to 131 pounds, but it increased during the few weeks before admission to 135 pounds. For 2 weeks preceding entry he noted a rash on the trunk and lower extremities, with trouserlike distribution. There was considerable pruritus associated with the rash. The patient had taken barbital and atropine at irregular intervals and, while at work, was exposed to acetone and latex rubber cement.

Physical examination showed a well-developed and nourished man in no acute discomfort. The skin was dry and shiny. There was a diffuse macular erythema which was slight on the upper extremities but which was more intense and girdlelike below the umbilicus and extended down both lower extremities. The lungs were clear. The heart was not enlarged, and there were no murmurs. The sounds were of poor quality. The blood pressure was not recorded. There were slight tenderness and spasm on deep pressure in the midepigastrium, more on the right of the midline. No masses were felt.

The temperature, pulse and respirations were normal.

Examination of the urine was negative. The blood showed a red cell count of 5,000,000, with a hemoglobin of 95 per cent. The white cell count was 11,600, 59 per cent polymorphonuclears. Stool examinations showed a 1+ reaction to the guaiac test on several occasions. A blood Hinton test was negative. Gastric analysis showed a free acid of 29 units and a combined acid of 10 units. A guaiac test on the gastric fluid was negative. The sedimentation rate was 0.85 millimeters per minute. An electrocardiogram showed a flat, slightly diphasic T_1. Q_4 was absent and T_4 slightly upright.

X-ray examination of the chest showed the diaphragm in normal position. The costophrenic angles were clear. The lung fields were not remarkable. The left ventricle was slightly enlarged downward and to the left. The aorta was slightly tortuous with a prominent aortic knob. The mediastinum was not displaced. Examination of the gastrointestinal tract showed considerable fluid in the stomach. There was an annular filling defect involving the pylorus and the pyloric end of the stomach, producing a concave defect in the base of the cap. Barium was retained in this area.

Shortly after the gastrointestinal series, the patient began to vomit repeatedly and Wangensteen drainage was instituted. Operation was advised, but the patient left the hospital against advice on the fifth hospital day.

Second Admission (2 months later). Following his discharge, the patient continued to have frequent attacks of vomiting associated with severe cramplike epigastric pain that caused him to double up. The vomitus was light brown and bitter. The pain had no relation to the ingestion of food; it occurred frequently at night and was only slightly relieved by soda. The dull ache in the right flank which the patient had complained of at the initial entry had subsided spontaneously. Pain in the lower back still occurred, however, and appeared to be associated with the cramplike epigastric discomfort. His bowel movements were regular but occasionally were chocolate-colored or tarry in appearance, usually following medication. Fatigue, weakness and malaise were progressive, but he had gained 5 pounds since his previous entry.

Physical examination showed no change from that previously noted. The blood pressure was 120/80.

The temperature was 98.8°F., the pulse 80. The respirations were 18.

Examination of the urine showed a red cell count of 5,200,000, with a hemoglobin of 95 per cent. The white cell count was 11,600, 29 per cent polymorphonuclears. The nonprotein nitrogen of the blood was 34 milligrams. Gastric analysis showed 13 units of free acid.

X-ray examination of the stomach showed narrowing and questionable ulceration of the prepyloric region. The cap was not deformed. Another gastrointestinal study showed moderate widening of the gastric rugae. There was

marked swelling of the prepyloric region over an area 5 centimeters in diameter. This region showed a small, constant narrowing through which peristaltic waves continued to pass. The base of the cap was concave.

On the third hospital day a gastroscopy showed normal peristaltic waves passing over the antrum to the pylorus, which was well seen and appeared to open and close normally. No ulceration or neoplasm could be seen, but on the lesser curvature, near the pylorus, there was a small polypoid excrescence, and the antrum appeared slightly redder than usual. The rugae of the greater curvature were rather large and tortuous, but after inflation they flattened out and appeared normal except for superficial reddening along their crests. The finding indicated a superficial gastritis.

On the fifth hospital day a laparotomy was performed.

Differential Diagnosis

Dr. Alfred Kranes: May I ask before starting to discuss this case whether this patient came to autopsy?

Dr. Benjamin Castleman: The patient did not die. We have only the surgical specimen.

Dr. Kranes: That helps us considerably for two reasons. In the first place, we will not have to worry about whether this patient had a coronary thrombosis, although the history and the electrocardiographic findings, particularly the T wave changes and the absent Q_4, are quite consistent with this diagnosis. In the second place, it will eliminate the necessity for discussing the possibilities of such things as gastric crises and lead poisoning as the cause of his symptoms, inasmuch as the pathological diagnosis of these conditions cannot be made. With these out of the way, we can concentrate on some local causes for the patient's symptoms.

The history is not characteristic of any particular disease. It certainly is not the story of an ulcer for a number of reasons. In the first place, the pain does not seem to bear any relation to food. Secondly, pain of the severity described here is rarely encountered in patients with ulcer unless the ulcer is penetrating, and of that we have no evidence. It does seem as though some pyloric obstruction was present, and from the history I do not believe one can safely go farther. If anything, I would lean more toward a diagnosis of gall bladder disease than toward any intragastric lesion.

Physical examination does not help us very much, nor does the routine laboratory work. I cannot understand why the sedimentation rate was elevated, as there does not seem to be any evidence of infection. I believe we shall have to forget about that. Certainly, the low-normal gastric acidity is not what one would expect to find with a peptic ulcer.

The important findings are confined to the x-ray examination of the gastrointestinal tract which seems to indicate a pathologic process of some sort in the region of the pyloric end of the stomach—a condition which would agree with the impression gained from the history of pyloric obstruction. It would have been very valuable if we had had some information as to the quantity of the fasting gastric contents, as this would be of further help in enabling us to say whether there was actual pyloric obstruction. The size of the stomach in the x-ray films, however, would not seem to support such a diagnosis. Usually the stomach is rather large and atonic in pyloric obstruction, whereas in the films shown here it seems to be of normal size. I think we can conclude, therefore, that, if pyloric obstruction was present, it was not chronic but rather of an intermittent type. The history of the interval following his discharge also seems to indicate either intermittent pyloric obstruction or biliary colic. The severity of the pain, as described here, would be very unusual for either a carcinoma or an ulcer of the stomach. The pain described in the back on both the first and second admissions is rather interesting and is quite consistent with an upper gastrointestinal lesion. Back pain may be the only symptom of a duodenal ulcer, for instance. We have recently had in this hospital several patients who have been thoroughly studied neurologically because of severe back pain and in whom a duodenal ulcer was found; treatment of the ulcer completely relieved the pain. How to explain it, I do not know; but it is an occasional finding in this type of case.

The gain in weight prior to both admissions to the hospital is distinctly against the presence of any malignant disease. Usually, patients with malignant disease, who lose as much weight as this patient apparently has, rarely regain any portion of it.

The laboratory work on the second admission does not help us any more than it did on the first, and the x-ray findings are, if anything, more confusing than ever. The gastrointestinal series seems to show something a little different from the previous one. I am rather disappointed that a Graham test was not done, as some of the patient's symptoms certainly suggest the possibility of a pathologic gall bladder with stones. I suppose the fact that a definite lesion of some sort was demonstrated on x-ray of the stomach made it unnecessary to study the patient any further.

In view of the facts that we have in this case, the differential diagnosis boils down to three possibilities: (1) peptic ulcer, (2) carcinoma of the stomach, (3) benign polyp. The evidence for ulcer is extremely meager. Certainly

this patient had none of the characteristic symptoms that we are accustomed to associate with a peptic ulcer. The x-ray picture is not that of a peptic ulcer, which is usually easily demonstrated roentgenologically. The x-ray reports are rather confusing and the picture seems to change, but there is no constant ulcer crater demonstrable. Moreover, I do not believe this patient would have lost so much weight from a peptic ulcer unless he had pyloric obstruction, and of that there is not much evidence. Finally, the very low gastric acid is definitely against a peptic ulcer. I therefore feel inclined to exclude this as a cause for his symptoms.

What about carcinoma of the pyloric end of the stomach? The x-ray described an annular filling defect, but this did not seem to be constant. Again, I would like to repeat that it would be extremely unusual for a patient to lose and then to gain weight with a carcinoma of the stomach. The absence of anemia, although it does not absolutely exclude carcinoma, is definitely against it. In addition, pain of this severity is rarely experienced in carcinoma of the stomach. The gastroscopic findings will have to be taken fairly seriously, as to my mind they explain a good deal of the picture. The polypoid excrescence might produce such a picture although its exact size is not clear from the description. If the patient had a pedunculated polyp near the pylorus, it could produce intense abdominal pain, nausea and vomiting by intermittent invagination through the pylorus. Certainly the character of the symptoms is more like that of intermittent pyloric obstruction than anything else; also such a diagnosis might explain the variability in the x-ray picture, as a long pedunculated polyp might move around and give changing pictures each time the patient was examined. In conclusion, therefore, I should say that ulcer and cancer are both rather unlikely in view of the story and that the best explanation I can give for this patient's symptoms is a pedunculated polyp at the pyloric end of the stomach, periodically invaginating itself through the pylorus and causing intermittent pyloric obstruction.

Dr. Edward B. Benedict: In concluding my gastroscopic report on this patient I said, "The findings indicate a superficial gastritis. No ulcer or carcinoma was seen. Because of angulation it is possible to miss certain areas along the lesser curvature near the pylorus by gastroscopy. In view of the history, presence of free acid, gain in weight, and gastroscopic appearance, I would favor a diagnosis of healing peptic ulcer with obstruction, rather than neoplasm. I believe, however, that the patient is temperamentally unsuited to follow a medical regime, and that surgery will probably be necessary." I realized at the time that "a small polypoid excrescence" may not be a true polyp but simply an enlarged fold. In omitting my conclusions from the history given to Dr. Kranes, the question of polyp was much overemphasized. My only postoperative diagnosis was superficial gastritis. Owing to peristaltic contraction with some spasm just proximal to the pylorus, the prepyloric ulcer could not be seen by gastroscopy.

Clinical Diagnosis

Hypertrophic gastritis.

Dr. Alfred Kranes's Diagnosis

Pedunculated polyp of the stomach.

Anatomic Diagnosis

Gastric ulcer.

Pathological Discussion

Dr. Benjamin Castleman: The operation consisted of a pyloric resection with a Billroth II type of posterior gastroenterostomy. Upon opening the specimen, we found in the immediate prepyloric region on the posterior wall a sharply demarcated ulcer approximately 1.5 centimeters in diameter and 0.5 centimeters in depth. The base was smooth and red, and the edges were soft. From gross examination we felt that the lesion was definitely on the gastric side, and in spite of the fact that a large percentage of ulcers in this region prove to be malignant histologically, the sharpness of its outline and its general appearance made us lean strongly toward a diagnosis of a benign ulcer. Microscopic examination confirmed our gross impression. The ulcer was fairly active and extended into the muscularis. The base was covered with a thick layer of fibrin. There was no epithelial atypicality at the margins of the ulcer, and after cutting serial sections, we were able to rule out malignancy in any part of the lesion.

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STUDY OF ALCOHOLISM

FINANCED by a grant of $44,000 from the Works Progress Administration, a research project that aims at a scientific study of the causes, costs and consequences of alcoholism has been launched at the Boston City Hospital. The committee in charge of the study consists of Dr. John A. Foley, chairman; Dr. Tracy J. Putnam, vice-chairman; Miss M. Geneva Gray, secretary and technician, and Dr. Merrill Moore, treasurer and director.

The reason for the study at this time is the conviction that the problem of alcoholism has grown larger and more complicated and, having been in the past treated as a political or a moral problem, has been neglected to a large extent by the medical profession. National prohibition—the political attempt to solve the problem—was an admitted failure, and many clergymen themselves now concede that more than a moral factor is involved. The magnitude of the problem is demonstrated by the fact that last year more than 5,000 persons were treated for alcoholism at the Boston City Hospital; many of them were repeaters, and there were a growing number of younger people.

By this study it is planned to gather all available statistics as to the number of alcoholics in the city in need of treatment, the cost to the city for their hospital treatment alone, and the cost to the public indirectly for the support of hospitals for the insane, social welfare and charitable organizations and other institutions made necessary in part by the effects of alcohol on the victims themselves and in part by the care of those in some way connected with or dependent on them.

Blood chemical tests will be utilized to determine how far alcohol is responsible for the condition of patients brought into the hospital in an unconscious state, and it is hoped that methods will be perfected or discovered that will hasten the sobering-up process without further damage to the system. It is expected also that information will be gathered—and this is of the greatest importance—concerning the mental and environmental conditions that lead to excessive drinking, with a view toward curing individual cases.

The committee estimates that its present funds will enable it to carry on the study until next month, and hopes that the importance of the work will by then have become so well recognized that further funds will be made available.

COMMENT ON THE AMERICAN FOUNDATION REPORT

EDITORIAL comment on the two volumes "American Medicine: Expert Testimony Out of Court", recently published by the American Foundation, has been so favorable—even in the columns of the publication representative of an organization whose officers have, in the past, refused to admit the need of changes in the methods and principles of medical care — that no doubt should exist as to the value of this material as an accurate expression of medical opinion.

One of the better editorials is that contained in the April fifteenth issue of the *New York*

State Journal of Medicine. This editorial comments upon the necessity for revision of our ideas regarding doctor-patient relations; upon the failure of any proposed compulsory health insurance plan to provide adequate care for the indigent and for those in rural communities and to consider the quality of medical care; upon the inadvisability of any scheme fostered and presumably dictated to by politicians; upon provisions for the proper medical care of the indigent; upon the protection of existing, philanthropically supported, medical institutions; upon the correlation of the measures of preventive medicine with the practice of the physician; and, finally, upon the feasibility, possibility and workability of integrating the private practice of medicine with the principles and ideas that are given. Possibly the medical supervision of community and state health problems is too little emphasized, but as a whole the editorial gives an extremely intelligent and ably expressed opinion on the broader aspects of these problems which are so important to the future of organized medicine.

In the belief that our readers will be interested in this comment, permission has been obtained from the copyright owners to reprint the editorial in toto.

American Foundation Report

To any one who reads the voluminous report of the American Foundation on its medical inquiry, it becomes apparent at once that the solutions for the problems inherent in the easy availability and delivery of medical care to the American people, as well as the sustained maintenance of high general health standards (which of course must include extension of preventive medicine) cannot be obtained by any easily devised short-cuts.

It is also evident that organized medicine must revise a few of its hitherto strongly held conceptions. A clearer comprehension of the implications of the doctor-patient relationship seems in order. The integration of the general practitioner with preventive palliatives seems warranted. The concept "that adequate medical care is, and always has been available to all the American people" needs further study and elucidation. Is it not true that we, as physicians, have too little personal contact with the "medically indigent" because we usually are not wont to meet them individually as our patients, but rather meet them as a class-group in our clinics and wards, because financial barriers hinder them from reaching us personally? Does doctor-patient relationship come before, or only after we meet them in our wards and clinics? They too need and want the factors in medicine which we as individual physicians could give them, to handle an incipient illness and prevent its progression toward serious consequences. It is in just such instances, that the availability of adequate medical care is hampered and hindered by financial barriers, and perhaps this is gradually becoming more important than the actual restoration to health of those who are sick. Here then, is another problem—how to provide high quality, easily available medical care to the medically indigent. But why stress details now.

Elsewhere in this issue will be found an abstract of the report. From the mass of evidence presented, there emerges an emphasis, as it were, *on ten outstanding principles.* To read these ten principles and comprehend their significant import is to sense the high quality of the contribution made by this report. We believe that it will evoke much discussion and provoke medical thought and perhaps action to the end that a start at least will be made toward solving some of the complexing questions of the instant problems.

We, of organized medicine, are now faced with an obligation to the profession as well as a duty to the public. We shall have to alter our general attitude. We cannot continue our hitherto "passive" position. We were fully justified to remain either "passive" or "in opposition" when the *only solution* tendered us was one comprising *compulsory health insurance.* It must be apparent even to the casual reader of this report that the problem is much larger, has greater implications, and must reach elements and factors in our population not even touched upon by the most ambitious compulsory health insurance scheme ever devised. Compulsory health insurance only touches upon the economic barrier separating the lower wage-earning groups from medical aid. It does nothing for the actually indigent, nor does it encompass any solution to the medical problems inherent to the situation of the rural and farming communities. It is totally unconcerned with the *quality* of medical care, and the many facets of the general medical problems which the report illuminates are left entirely unapproached to say nothing of even making a crude attempt to solve them. The entire question of preventive medicine is not even envisaged in compulsory health insurance schemes.

As a unit of organized medicine there is another consideration to which we must give thought. This is the political implication of the present situation in Washington. Were we, unfortunately, to have compulsory health insurance foisted upon us at this time, while the other elements and factors of the larger and more widespread questions remain unprovided for, unanswered and unplanned for, this very much larger field—revealed by the report as demanding serious consideration—would remain for a long time uncorrected, without intelligent planning, and without even an honest effort at seeking solutions for its problems. This would happen because there would result a complete cessation of all further endeavors to meet the medical issues of larger and more significant import. *Compulsory health insurance must be stopped while we, the organized profession, take the necessary positive steps to meet squarely these greater issues.* We know that all further efforts aiming at health standards, and the delivery of a high quality of medical care, would cease because legislators, having enacted the health insurance laws with their inevitable tax burdens, would conceive that they had "done enough for health." Government instead of setting out to study and evolve solutions to the greater medical questions raised by this report, would be too busy and too concerned in establishing the necessary bureaucracy to make compulsory health insurance work, in the face of a recalcitrant medical profession only half-heartedly assisting to establish a scheme of delivering medical care which it knows beforehand will reduce its quality; and will actually sell the population a "gold brick" for the money paid in direct, indirect and hidden taxes. And what about the medical institutions which produce doctors—the laboratories, the wards, and the operating rooms, — the workshops of the profession, — their

financial status rendered progressively more precarious because of the failure of private endowments and awards to support them, would find existence increasingly difficult. There is a very paramount issue therefore facing us now. We must perforce take a positive stand. The report of the American Foundation spreads opinions before us on the many complexities of the problem. From the study of these opinions we must have proposals ready which must encompass the whole field now opened to our scrutiny. When we have further studied this report, let us formulate a proposal upon which government, the profession, and the enlightened public can unite.

As we study this report further it becomes evident that the health of the community, as a whole, is a more paramount issue of the state than it is a problem of physicians whose first concern is the individual. Organized medicine can determine standards, and decide qualifying conditions under which the best of medical care may be obtained. It naturally must be the concern of the State to see that this high quality of medical service is easily obtainable by the citizenry. We have consistently held that the indigent are properly the charge and wards of the local communities. This report repeats this stress. We have contended that the medical care to these must necessarily be a charge against funds raised by taxes, and the physician treating these indigents be paid so that the doctor serving the indigents is not twice taxed while his lay neighbor pays only one tax for the same purpose.

Of the "medical indigent" we have already spoken above. They need education to teach them *how to reach the doctor* when financial barriers seem to bar the way. This too is a local community problem more than one confronting nationally organized medical units. The community must establish agencies to arrange the finances necessary to pay the charges incurred by the "medically indigent" to cover the physician's services and the hospital costs. Perhaps some variation of the so-called DeSanctis plan holds the answer to this problem. This entails the establishment of a locally supervised bureau to determine what these medically indigent shall pay toward the discharge of their obligation, and yet it seems to us, that the local community too must contribute toward the delivery of high quality medical care to this underprivileged class-group. The rate of fees which the physician shall charge, to make the best services available to this group, is another problem facing organized medicine. In the cooperative era upon which we are entering, such planning is not only in proper order but urgently necessary.

Since taxation in the present era is depleting private philanthropy, state aid through Federal grants will soon become necessary if we are to maintain and sustain many existing fine medical institutions including hospitals and laboratories.

If we desire to meet the instant problem presented by the practitioners in rural and outlying areas, and in sparsely settled communities, laboratory and consultative services must be made available to the country general practitioner. Can such laboratories and medical consultative service exist without governmental aid?

Finally, it is very evident that a large and more active evolutionary development of preventive medicine is impending. We would welcome this were we to see it worked out so that the private practitioner would actually become the health official who would deliver the palliatives which medical science has won for the benefit of better health for the community. In the evolution of this larger health program,—a program which must envisage sanitation and hygiene and reduce health hazards in field, mine, factory, school, store, and home,—it would seem almost axiomatic that there must be established *a Federal Department of Health*, under a competent physician, separate and apart from all other welfare agencies. This item is now pending in *our national* legislature.

Finally we conclude from our perusal of the report, as the problems are touched upon by our leading medical men, that the private practice of medicine shall continue, and that it is feasible, possible, and workable to integrate this private practice of medicine with the ideas and principles here but briefly sketched.—*New York State Journal of Medicine* 37:730 (Apr. 15) 1937.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

ELLIS, LAURENCE B. M.D. Harvard Medical School, 1926. Instructor in medicine, Harvard Medical School. Junior visiting physician, Boston City Hospital. Assistant physician, Thorndike Memorial Laboratory, Boston City Hospital. Address: 319 Longwood Avenue, Boston. His subject is "The Treatment of Bright's Disease and Related Renal Infections." Page 821.

MILLER, RICHARD H. M.D. Harvard Medical School, 1910. F.A.C.S. Visiting surgeon, Massachusetts General Hospital. Clinical professor of surgery, Harvard Medical School. Address: 264 Beacon Street, Boston.

SMITH-PETERSEN, M. N. M.D. Harvard Medical School, 1914. Chief of the Orthopedic Service, Massachusetts General Hospital. Clinical professor of orthopedic surgery, Harvard Medical School. Address: 264 Beacon Street, Boston. Their subject is "Further Report on Osteomyelitis at the Massachusetts General Hospital." Page 827.

ROBINSON, LEON J. M.D. Boston University School of Medicine, 1934. Formerly, house officer, Fifth Medical Service, Boston City Hospital. Resident in epilepsy, Monson State Hospital. Now, assistant physician, Monson State Hospital. Address: Palmer, Mass. His subject is "Neurologic Complications Following the Administration of Vaccines and Serums: Report of a Case of Peripheral Paralysis Following the Injection of Typhoid Vaccine." Page 831.

ADAMS, JOHN D. M.D. Harvard Medical School, 1902. F.A.C.S. Assistant professor of orthopedic surgery, Tufts College Medical School. Instructor in orthopedic surgery, Harvard Graduate School of Medicine. Orthopedic surgeon-in-chief, New England Medical Center. Consultant orthopedic surgeon, Jordan Hospital, Plymouth; Lawrence General Hospital, Lawrence; Beverly Hospital, Beverly; and Melrose Hospital, Melrose. Address: 43 Bay State Road, Boston. His subject is "A Report on Six Cases of Supracondylar Fractures of the Elbow." Page 837.

The Massachusetts Medical Society

ENTERTAINMENT OF LADIES AT THIS YEAR'S ANNUAL MEETING

THOSE of us who attend the Society's annual meetings look forward to the social aspect of this pleasant interval in our daily routine as much —or almost as much—as to the more serious purpose of our coming together. For the ladies of our families, however, this distressing duality of purpose need not exist, since our very efficient Ladies' Committee has completed plans guaranteed to result in a thoroughly enjoyable three days, allowing practically no time for shopping or any other serious pursuit.

On Tuesday afternoon a tea will be given in Brookline by Mrs. Charles Mixter. That evening comes the Shattuck Lecture. Wednesday morning will be devoted to visiting hospitals, with a tea and reception at the Gardner Museum in the afternoon. On Wednesday evening during the Annual Dinner, the ladies will dine in the Penthouse, Hotel Bradford, where a special floor show will be put on for them, after which the speeches in the men's dining room may be attended. There will then be dancing to take care of the excess energy anyone may have. At ten o'clock Thursday morning busses will leave for the Wayside Inn in Sudbury, where there will be a luncheon to meet the wives of the district presidents. The survivors will be returned to the Hotel Bradford in Boston by three in the afternoon.

Those who are planning not to take in the annual meeting this year are frankly advised to hide this program from their wives.

SECTION OF OBSTETRICS AND GYNECOLOGY*

R. S. TITUS, M.D., *Chairman*, 472 Commonwealth Ave., Boston, Mass.
R. J. HEFFERNAN, M.D., *Secretary*, 1101 Beacon St., Brookline, Mass.

CASE HISTORIES

No. 19. INTERSTITIAL PREGNANCY

Mrs. F., a white woman, 36 years old, entered the emergency ward of the Massachusetts General Hospital, January 28, 1935, with a presumptive diagnosis of incomplete miscarriage.

Her family history was essentially negative. Her father was living and well. Her mother had died at 76 of senility. There was no tuberculosis, cancer or diabetes in the family history. She had had mumps and measles. Her tonsils and adenoids had been removed in 1910. She had four children, living and well, the last one having been born 8 years previously. Prior to the birth of her first child she had had chorea. Her periods began at the age of 15 and were always regular, lasting 4 to 5 days, with a 28 day interval. Her last period was October 15, 1934.

On entrance to the hospital, the patient's temperature was 99.2°F., her pulse 88 and her respirations 24. Her blood pressure was 110/86. Her mucous membranes were of normal color. Her teeth were poorly cared for and showed some evidence of pyorrhea. Her heart was not enlarged, and there were no murmurs. Her lungs were resonant throughout, and there were no rales present. The abdomen was soft, with no evidence of masses and no tenderness. Vaginal examination revealed a multiparous perineum, a bilaterally torn cervix, which seemed firm, and a fundus with a definitely enlarged irregularity in contour and a questionable mass in the right cornual region.

The patient's red blood cell count was 4,200,000, the white cell count was 8,750, and the hemoglobin 80 per cent (Tallqvist). A specimen of urine showed no albumin and no sugar, and the sediment contained no abnormal constituents. An Aschheim-Zondek test was performed on January 30, and was positive.

The patient said that 3 days after her first missed period, she had made several attempts to induce an abortion. These attempts were followed by a small amount of staining, which continued off and on from November 17 until about the first of December. On December 18 she started to flow and flowed continuously until December 27. At that time, she was put to bed for a couple of days by her local doctor because the flowing was so profuse. On January 13 the staining reappeared, and on January 20 she again began to flow profusely, passing clots. On entrance to the hospital there was only a small amount of actual bleeding. From the history and from the size of the uterus a diagnosis of incomplete abortion with a question of fibroids was made.

Under ether anesthesia, the uterus was curetted. No material was obtained, but the mass on the right was definitely made out. The abdomen was opened; the mass in the right cornus was described as cystic and dark in appearance, and it looked as if it contained old blood. It had not ruptured because there was no evidence of blood in the abdominal cavity. A supravaginal hysterectomy was done, and the patient made an uneventful convalescence.

The diagnosis of interstitial pregnancy was considered at the time of operation. The following is the pathological report: "A normal-shaped uterus, approximately 8 centimeters in length, which has been opened longitudinally to expose a normal-appearing myometrium, 2 cen-

*A series of selected case histories by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

timeters in thickness, and a fairly dry, pink endometrium. At the fundus is a circular, dark red, roughened area covered with blood clots. It constitutes what seems to be an interstitial or subserous, disk-shaped mass, 1.5 centimeters thick. There is only a layer of peritoneum covering the external surface. On section through the mass, it seems to be attached at one place to the endometrium in the isthmus. The cervix is not present; the internal os has been cauterized. There is also a moderate amount of curettings. Diagnosis: isthmal pregnancy."

Comment: A diagnosis of interstitial pregnancy cannot be made until the abdomen is opened. The presence of a mass with irregular flowing might suggest a fibroid complicating pregnancy or an ectopic pregnancy. It is possible that, if the diagnosis had been thoroughly appreciated at the time of operation, the pregnancy might have been evacuated by hysterotomy, thereby leaving a perfectly good uterus.

MISCELLANY

THE GENERAL PRACTITIONER AND TUBERCULOSIS

In any tuberculosis control program the general practitioner is the connecting link between the patient and the tuberculosis specialist. Each of the three has certain responsibilities regarding the early diagnosis of tuberculosis and each, therefore, must bear some of the blame for cases diagnosed late — the patient in that he has disregarded his symptoms, the practitioner in that he did not "tumble to it earlier," the tuberculosis specialist for failing to x-ray suspected cases. How may this chain be strengthened?

Considering, first, the link between the patient and his doctor, it is patently impossible to set the machinery in motion unless the patient comes to his doctor. Can anything be done to reduce the quite considerable percentage of patients who ignore their symptoms? The answer is, of course, a more adequate health-education program so that "the public can be better informed about the onset of tuberculosis and the importance of early diagnosis." And the responsibility rests on tuberculosis associations and health departments.

In other cases, however, the delay in diagnosis is chargeable not to the patient but to the physician. Here medical education must bear a large part of the blame. All too often the cases of tuberculosis seen by the medical student show all the classical signs and are, therefore, in an advanced stage. It is difficult, too, for the student to appreciate the slowness of the clinical course of tuberculosis. Most illnesses can be watched by the intern from immediately after onset until their termination. In tuberculosis he has "brief glimpses of different individuals at different stages and with different reactions" and "he is a genius if he can piece together the story from those odd chapters torn from different books." In this instance the challenge is to the medical schools.

Yet, whatever the shortcomings of his undergraduate training in the diagnosis and treatment of tuberculosis, the physician who engages in general practice will encounter many cases of tuberculosis and must therefore keep it ever in mind and use every opportunity for improving his ability to diagnose it. The author would impress upon the minds of all newly qualified doctors the following two thoughts:

"Always be on the lookout for tuberculosis even though you run the risk of being thought a crank for your pains. Never label an illness 'influenza' if there is no epidemic without subsequently having the chest x-rayed to make sure."

The general practitioner will frequently encounter a case which he has reason to believe is tuberculosis, but on which he needs expert advice or additional diagnostic facilities; it should be possible for him to secure x-ray plates of his patient's chest and where necessary the services of a tuberculosis specialist.

He is also much more likely to maintain his zeal for early diagnosis if he knows that adequate provision is made for the treatment of his patient in the event that his suspicions prove to have been well founded. In the event that no facilities exist for the hospitalization of the tuberculous, he is likely to take the attitude that early diagnosis is of purely academic interest. To quote the author's analogy, — "supposing that neighboring surgeons would not operate on acute abdomens, one's zest for differentiating between perforated gastric ulcers and colic would be blunted, and one would fall too readily into the easy way of medicine which is —expectant treatment and guarded prognosis."

The general practitioner occupies a position of great strategic importance in the control of tuberculosis. He brings the patient into the world. On him in most cases devolves the responsibility for the initial diagnosis of tuberculosis and "after the tumult and the shouting have ceased, the tuberculosis officer and the sanatorium physicians departed, he tends the patient for the rest of his life and eases his parting."

The patient returns to him from the sanatorium a changed man, both physically and psychologically. Various things have happened. "First, he has come to accept the fact that he has tuberculosis and that it is a long complaint. Secondly, he has lived among other patients who are in the same plight. Thirdly, he has learned a lot about his complaint."

The general practitioner is now "the man on the spot." Yet much can be done to make his task easier and here are some of the ways in which the link between the general practitioner and the specialist (sanatorium physician) can be strengthened.

"I consider it a blasphemous act for anybody to undermine whatever faith a patient may have in his doctor. For the patient, it is better to have faith in a mediocre doctor than a distrust of the

best. For the average mediocre doctor it is far greater incentive to do good work and make himself worthy of the patient's unswerving (and often pathetic) belief in him. This is a matter in which tuberculosis officers, consultants and sanatorium physicians have the greatest power and where their closest cooperation is needed."

Secondly, a commission should be appointed to decide on postsanatorium advice and treatment. The points which they consider of value should be embodied in a small printed pamphlet of instructions capable of alternate readings dependent on the mere stroke of a pen. It should be a simple matter to draw up such a set of instructions. If one copy be given to the patient on discharge, another to his doctor and another to the tuberculosis officer, everybody would know where they were. In addition "I should like to see the doctor receive a copy of the x-ray photographs as well as the usual statistics such as body weight, blood sedimentation, rales, etc., which he can continue to record should he feel so inclined." Could a general practitioner read chest x-ray plates? Speaking for his late self of general practice days, the author's answer is—"No, he couldn't, but he would if he were given a chance."

Lastly, let the tuberculosis officer meet the doctor periodically for a real consultation over their shared cases. Once every 3 months say, there could be set aside a tuberculosis afternoon, when the tuberculosis officer would meet the general practitioner and their ambulant patients at the doctor's consulting room, after which they would proceed together to the homebound cases.

Such close cooperation would have in the author's opinion a most stimulating effect. "With it," he writes, "the alleged ignorance and carelessness of general practitioners could not exist. It is the sort of help I wanted and could not get. It is not an impossible demand for I know that in some localities it is in existence and works well. Could it not be made a universal practice?"

The campaign against tuberculosis requires a chain of cooperation the links of which should be equally strong. These links are those between the patient and the general practitioner and between the general practitioner and the tuberculosis specialist. Their respective responsibilities have been discussed by an author who has had extensive experience in both of the medical roles. His suggestions merit serious consideration.—*Tuberculosis Abstracts 10: May, 1937.*

The article referred to is one by Dr. George H. Day entitled "The general practitioner and tuberculosis" (J. State Med. 45:37 [Jan.] 1937).

MIDDLESEX COLLEGE APPOINTMENT

The Committee on Faculty at Middlesex College has recently announced the appointment of Dr. Israel Kahalas of 483 Beacon Street, Boston, to a didactic and clinical instructorship in the subject of pulmonary diseases in the School of Medicine. Dr. Kahalas, who resides at 40 Kirkwood Road, Brighton, is a graduate of Tufts College Medical School and was admitted to practice in Massachusetts in 1931. He interned at the Monmouth Memorial Hospital, Long Branch, N. J., and for five years was a resident physician at the Rutland State Hospital. He is a member of the staff of the Beth Israel Hospital, the Jewish Tuberculosis Sanatorium, Rutland, and the Out Patient Department of the Massachusetts General Hospital.

HOSPITALS BENEFICIARIES

The Massachusetts General and the New England Deaconess hospitals have been named as the principal beneficiaries of the estate of William A. Sargent, of Brookline, who died recently. Each of these institutions will receive a legacy of more than $1,000,000 under the terms of Mr. Sargent's will.

DR. MINOT HONORED

Dr. George R. Minot, of Boston, was recently elected a member of the National Academy of sciences. This organization is composed of distinguished Americans in the field of Science and is limited to 300 members.

CORRESPONDENCE

PRONTYLIN AND PRONTOSIL

April 26, 1937.

Editor, *New England Journal of Medicine,*

Following are two brief case reports that may be of interest to your readers:

The first case was that of an adult with a septic mastoid. The white blood cell count was 18,000, with 85 per cent polymorphonuclears; repeated counts gave approximately the same result. After giving prontylin and prontosil for several days, the white blood cell count dropped to 4,000, with 35 per cent polymorphonuclears. Withdrawal of the drugs was followed by high total and polymorphonuclear counts.

The second case was that of a primipara, who, after a stormy delivery, ran a febrile course with temperatures ranging from 104 to 105° F. The white blood cell count was 20,000, with over 80 per cent polymorphonuclears. After a few days, prontylin and prontosil were given, and the total count fell to 6,000, with from 50 to 60 per cent polymorphonuclears. The fever continued for 6 weeks, during which time the temperatures ranged from 101 to 103° F. and the blood counts, made almost daily, hovered between 5,000 and 6,000, with about 50 per cent polymorphonuclears; two transfusions were given. By this time a bacterial endocarditis was suspected, but the blood cultures remained sterile. Withdrawal of the drugs was promptly followed by a rise in the total count to 13,000, with over 80 per cent polymorphonuclears. A mild leukocytosis continued for 2 weeks, and the patient recovered.

As no other drugs were used in these cases, it may be assumed that the low blood counts were due to prontylin and prontosil.

Very truly yours,

A. TRUMPER, M.D., *Pathologist.*

St. Margaret's Hospital,
Montgomery, Alabama.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
May 4, 1937.

Managing Editor,
The New England Journal of Medicine,

In addition to the articles enumerated in our letter of April 6 the following have been accepted:

Eli Lilly & Company
Merthiolate Suppositories 1:1000

Medical Arts Laboratory
Rabies Vaccine (Killed Virus) seven 2 cc. vials package

National Aniline & Chemical Co., Inc.
Scarlet Red Sulphonate—"National"

Schering Corporation
Ampoule Solution Neo-Iopax, 10 cc.

Sterisol Ampoule Corporation
Sterisol Ampoules Dextrose 2½% in Physiological Solution of Sodium Chloride
Sterisol Ampoules Dextrose 10% in Physiological Solution of Sodium Chloride
Sterisol Ampoules Dextrose 20% in Physiological Solution of Sodium Chloride
Sterisol Ampoules Dextrose 25% in Physiological Solution of Sodium Chloride
Sterisol Ampoules Dextrose 5% in Distilled Water
Sterisol Ampoules Dextrose 10% in Distilled Water
Sterisol Ampoules Dextrose 20% in Distilled Water
Sterisol Ampoules Dextrose 25% in Distilled Water

The following article has been accepted for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Nonofficial Remedies, 1936, p. 471):

Sharp & Dohme
Tablets Theobromine with Sodium Salicylate

PAUL NICHOLAS LEECH, *Secretary.*

RECENT DEATHS

PRESCOTT—WILLIAM HERBERT PRESCOTT, M.D., of 563 West Central Street, Franklin, died at his home on May 5.

Born in Concord in 1860, Dr. Prescott attended the schools of Concord and was graduated from the Harvard Medical School in 1888. In the following year he was appointed a house officer at the Boston City Hospital and eventually became assistant superintendent of that institution.

He served as assistant in pathology and clinical medicine at the Harvard Medical School and, in 1896, was appointed director of public institutions in Boston. During the Spanish War he was an agent of the Massachusetts Volunteer Aid Association and an acting surgeon of the United States Army.

He was a retired member of the Massachusetts Medical Society and the American Medical Association and a member of the New England Society of Psychiatry and the Masonic Order.

His widow, Mrs. Kate H. Prescott, a son, Robert B. E. Prescott, of Seattle, Washington, and three daughters, Mrs. A. G. Churchward, of Glendale, California, Mrs. H. B. Kaster, of Berkeley, California, and Mrs. Jones, of Franklin, survive him.

CALL—EMMA L. CALL, M.D., of 19 Garden Street, Cambridge, died at the New England Hospital for Women and Children on May 3. She was in her ninety-first year.

A native of Newburyport, Dr. Call attended the public schools of Boston and studied medicine at the University of Michigan Medical School, where she received her medical degree in 1873. She studied in Vienna for a time before coming to Boston, where she practiced until her retirement twenty years ago.

She was one of the first woman physicians in the United States and the first woman to become a member of the Massachusetts Medical Society. She was also a member of the American Medical Association and at one time was attending physician and consultant in obstetrics at the New England Hospital for Women and Children.

NEW—WAY SUNG NEW, M.D., one of China's outstanding surgeons, died in Shanghai on May 4. He was in his forty-sixth year.

A graduate of the Harvard Medical School, Dr. New was surgeon at St. Luke's Hospital in New Bedford from 1914 to 1915 and later served at the Carney and Massachusetts General hospitals, of Boston, and the Johns Hopkins Hospital of Baltimore.

He was a member of the Massachusetts Medical Society and the American Medical Association.

REPORTS OF MEETINGS

BOSTON PATHOLOGICAL SOCIETY

The Boston Pathological Society met in the Pathology Department of the Massachusetts General Hospital, February 5. Dr. J. Beach Hazard presided. The first paper of the evening was presented by Dr. Robert S. Palmer and Dr. Benjamin Castleman on "A Tumor of the Adrenal Medulla Associated with Paroxysmal Hypertension." Dr. Palmer gave a brief summary of the clinical history of the case.

A 23 year old, white female entered the hospital complaining of headache. For 9 years she had experienced paroxysmal attacks of headache and for 4 years had been subject to fainting attacks, which were characterized by a choking sensation and by throbbing sensations in the chest and head. Physical examination showed extreme choking of both optic disks, marked eyeground changes and a blood pressure varying between 180 and 200 systolic, and 120 diastolic. The heart was not enlarged. The concentrating ability of the kidney was decreased. The attacks of fainting were precipitated on the wards by excitement, emotion or nervousness, and it was noted that during such attacks there were slowing of the pulse, dilatation of the pupils, generalized vasoconstriction, hyperglycemia and glycosuria. Because of the absence of localizing neurologic symptoms and the variety of symptoms presented, it was believed that the presence of a brain tumor was unlikely. It was suggested that a paraganglioma might be causing the symptoms, and an exploratory operation was planned. Before such a procedure could be carried out, however, the patient died during an attack of hypertension.

Paragangliomas are associated with symptoms similar to those seen in the present case, namely, symptoms of hyperadrenemia. They usually occur in young persons, and there is persistent hypertension between the paroxysmal attacks. The differential diagnosis between the paroxysmal hypertension due to such a tumor and the hypertensive crises observed in essential hypertension is extremely difficult.

There are two other types of adrenal tumor which are associated with attacks of extreme hypertension. The first of these types occurs characteristically in young individuals. There is a normal blood pressure between attacks, and there are no heart, kidney or eyeground changes. The second type occurs in patients that have not been known to have had previous attacks of hypertension. Such patients are apt to die suddenly after delivery, operation or accident, and the adrenal tumors are found at postmortem examination. Some 70 cases of all three types have been reported in the literature.

Dr. Palmer emphasized the harm that might result if every case of paroxysmal hypertension was explored surgically in an attempt to remove an adrenal tumor. He also stated that injection of air into the region of the adrenals, and subsequent x-ray studies were unlikely to prove the existence of such tumors, since they are usually situated in regions not visualized by this technic.

Paragangliomas are not to be confused with tumors of the adrenal cortex. The latter tumors cause syndromes of glandular dyscrasias, while the former are not associated with any alteration in other endocrine glands.

Dr. Castleman presented the pathologic findings in the case described by Dr. Palmer. At necropsy a large tumor was found in the region of the right adrenal. This tumor weighed 45 gm., was hard and fibrous and was surrounded by a narrow rim of adrenal cortical tissue. Microscopic examination of the tumor cells showed them to be large, atypical and extremely variable in size. Mitoses were numerous, and some cells had two or three nuclei. No chrome material was seen in the cytoplasm. There was some tendency toward alveolar formation. The tissue was assayed for adrenine content, and by computation it was found to contain a total of 15 mg. of this hormone. The normal adrenal contains a total of only 4 mg. Since this assay was not made until 20 hours postmortem, it is probable that much of the adrenine had been destroyed. There are several reports of similar tumors from which adrenine has been isolated in high concentration. Tumors of this type are known as pheochromocytomas.

The second paper, "The Application of Supravital Staining to the Study of Normal Lymphocytes and Those of Infectious Mononucleosis," was presented by Dr. Edward A. Gall. A brief description of the technic of supravital staining was given. Freshly drawn blood is placed on a coverslip that is coated with a thin, dry film of janus green or vital red. The cytoplasmic granules are stained, but not the nucleus. There is no motility of the cell when janus green is used but, if vital red alone is applied, the leukocytes are actively motile. By a study of the cell motility and the distribution of cytoplasmic granules, cells of the monocytic and lymphocytic series can be differentiated with ease. The monocyte moves by means of projecting a thin surface film of cytoplasm, which is followed by the nucleus and the remainder of the cell. The mitochondria in these cells appear as fine granules scattered throughout the cytoplasm which are stained by the janus green. Neutral red shows uniform-sized granules, which stain a brick-red hue and which may be segregated in clumps, although the rosette formation that occurs in rabbits and that was described by Sabin is rarely seen in human cells. The lymphocyte is characterized by a motility in which the nucleus is in the anterior portion of the cell and precedes the mass of cytoplasm. The cytoplasm is homogeneous and glassy in appearance, while the mitochondria are larger than those of the monocyte and show a tendency to form clumps in the vicinity of the nucleus. Granules stain a bright red with neutral red and are more refractile than those of the monocyte. In some 36 per cent of the lymphocytes of the circulating blood, there are one or two large, very highly refractile granules.

These large granules were not seen in more than 15 per cent of the cells of a series of bloods from 17 patients with infectious mononucleosis. The characteristic cells of this disease were found to be somewhat larger than normal lymphocytes, their nuclei were reniform in shape, and their cytoplasm was similar to that of the lymphocyte, with a smooth border. Their motility was the same as that of the lymphocyte. The mitochondria were coarser; they were scattered irregularly through the cytoplasm and not clumped around a "Hoff." The granules stained

like those of a lymphoblast and were often arranged in a loose circle around the nucleus. Dr. Gall suggested that supravital studies of blood might be of diagnostic value in questionable cases of infectious mononucleosis.

Dr. Castleman spoke on "Parathyroid Hyperplasia in Chronic Renal Failure." Enlargement of the parathyroid glands may be of three types: a parathyroid adenoma, usually involving one gland; a true neoplasia; and a diffuse enlargement of all glands, a true hyperplasia. The latter is of two types: a primary hyperplasia in which the cells are large with a water-clear cytoplasm, and in which the hyperplasia is believed to be caused by some as yet unrecognized hormonal imbalance; and a secondary hyperplasia, which occurs in chronic renal disease, and in which the cells are normal-appearing but greatly increased in number.

In cases of so-called primary hyperplasia, the level of the blood calcium is elevated, while the blood phosphorus compounds are decreased. Plasma phosphatase is increased, and histologic examination of the bone shows the characteristic changes of osteitis fibrosa cystica. In contrast, cases of hyperplasia due to chronic renal disease show a marked elevation of blood phosphorus and usually a decrease in blood calcium. The plasma phosphatase is elevated, and the bones may show a true osteitis fibrosa, which cannot be differentiated from that of primary hyperplasia. The necropsy findings in a case of advanced chronic glomerulonephritis were described. The blood calcium was found to be 10 mg. per cent, the plasma phosphorus 7.92 mg. per cent, and the plasma phosphatase 9.36 Bodansky units. There were marked calcification of the arteries and masses of calcium in the fingers and ilium. The parathyroid glands weighed 10 gm. and differed in appearance from the parathyroid glands of primary hyperplasia, which are soft, compressible and orange-brown in color. The glands in this case were very firm and grey in color. Histologically, they were composed of increased numbers of normal-appearing chief cells, showing a tendency toward acinar and papillary arrangement. There were no mitoses, and there was an absence of fat cells, which are normally found lying between the cells of the parenchyma. An increased number of oxyphil cells were present. The bone marrow was replaced by fibrous tissue, and there was a marked increase in osteoblasts and osteoclasts.

It is believed that the chief cells themselves do not produce a hormone, and that the increase in their numbers represents an increased demand of the body for the secretions of the more mature cells, with a regenerative response by the gland and a hyperplasia of the chief cells in an attempt to produce more oxyphil cells.

A study of the parathyroid glands from routine necropsies revealed some degree of hyperplasia in almost all cases of chronic renal disease. There was also hyperplasia in certain cases of bone disease, such as rickets, Paget's disease and metastatic carcinoma of the bones.

The retention of phosphorus compounds because of renal insufficiency is believed to be the cause of the hyperplasia. Hyperplasia of the parathyroid glands has been produced in rabbits by repeated injections of phosphorus compounds.

Dr. Mallory stated that hyperparathyroidism itself could produce chronic renal failure because of the formation of small calculi in the loops of Henle with injury to the renal parenchyma. Since secondary osteitis fibrosa develops in advanced renal disease, it may be impossible to differentiate the two conditions clinically, although examination of the parathyroids will show a specific picture in each condition.

The last paper of the evening, "Clinical and Pathologic Aspects of Bronchial Adenomas," was presented by Dr. Donald S. King and Dr. Tracy B. Mallory. Dr. King reviewed the symptomatology of 12 cases of benign adenoma of the bronchus. The chief symptoms, in order of their importance, were found to be: dry cough, hemoptysis, pleural pain with the onset of sepsis, fever with chills and sweats, dyspnea and wheezing. Physical signs of atelectasis below the obstructing adenoma are frequently found. Bronchiectasis distal to the adenoma may or may not be present. Dr. King advised bronchoscopic removal of the tumor rather than lobectomy or pneumonectomy, unless the lung below the obstruction is markedly diseased.

Dr. Mallory stated that, although benign adenoma of the bronchus is a definite clinical and pathologic entity, the exact type of cell from which it arises is unidentified. Such tumors characteristically occur in young persons, usually between the ages of 20 and 30, and are invariably located in one of the major bronchi or in the lower trachea. They grow into the lumen of the bronchus and also outward between two bronchial cartilages, forming a dumbbell-shaped tumor, one half of which lies within the bronchus, and one half outside. Histologically, such tumors may appear to be malignant on casual inspection, and many pathologists have called them bronchiogenic carcinomas. No mitotic figures are found, however, and there have never been metastases from these tumors. Occasionally they may resemble basal cell carcinomas or carcinoids, and spicules of bone or calcium are frequently found in their substance. There is no gland formation, mucous secretion or tendency to differentiation, although there may be pseudoglandular formation around sinusoidal blood vessels. Such tumors apparently grow upwards from the submucosa, pushing the normal mucosa before them, and there is a marked tendency for superficial ulceration, so that bronchoscopic biopsies frequently consist only of mucosa showing inflammatory reaction. Dr. Mallory emphasized the clinical importance of recognizing such tumors, and stated that bronchoscopic removal seemed the logical way of treating them. The prognosis in such cases is very good.

GREATER BOSTON MEDICAL SOCIETY

The Greater Boston Medical Society met December 1, 1936, in the auditorium of the Beth Israel Hospital to hear a series of papers arranged as a "Symposium on Abdominal Pain in Pediatrics." Dr. Hyman Green served as chairman and introduced the speakers.

Dr. Harold Freedman discussed the "Relationship Between Upper Respiratory Infection and Associated Abdominal Pain in Children." He stated that youngsters do not complain of sore throat as often as they do of pain in the abdomen when they are suffering from respiratory infections. The abdominal pain is usually representative of one of two main types. In the first type, the pain is constant or colicky and is referred to the umbilicus; there is no tenderness, but usually a certain amount of voluntary spasm, which disappears when the child is asleep. Rectal examination in such cases is negative. In the second type, the pain is constant and often has associated tenderness; it is referred to the umbilicus, right side or right lower quadrant and is apparently derived from the involvement of the mesenteric and retroperitoneal glands, with or without associated lymphangitis. Since pyelitis is a frequent complication of respiratory infections, particularly in females, a urinalysis should always be done. An onset of pain with chills and fever is suggestive of pyelitis. When intrathoracic conditions, such as pneumonia, pleurisy, pericarditis and pleurodynia, present themselves with abdominal pain, it is possible to tell that the process is in the thorax from the character of the breathing and the general appearance of the patient. The physical examination is usually more helpful than the knowledge of the pulse rate, fever and leukocytosis in deciding the differential diagnosis. Since abdominal conditions demanding surgical attention may occur in the presence of an upper respiratory tract infection, a rectal examination should never be omitted.

Dr. George Kahn discussed the "Medical Causes of Abdominal Pain" and stated that in infants it is hard to tell about abdominal pain except from the postural reaction and from distention. Although "colic babies" may prove very distressing problems, it is easy to stop the commonest causes of colic, such as hunger, constipation, too much air swallowing, diarrhea and overhandling, and, thus, to cure the colic. Acute indigestion may be differentiated from appendicitis because abdominal tenderness is absent or diffuse; chronic indigestion, a rare condition, is due to intolerance toward certain dietary elements. A sensitive and easily stimulated bowel may be stirred to hypermotility by vegetables or fruits and give rise to pain. Acute constipation produces diffuse lower abdominal pain, more especially on the left side, with palpable fecal masses; the treatment is catharsis after acute appendicitis has been ruled out. Chronic constipation results from poor bowel habits and is corrected by the institution of proper bowel hygiene. When constipation is due to the absence of roughage, the stools at first are marble-sized, but later on, an atonic, dilated bowel forms large-caliber stools. Among the rarer causes of abdominal pain in childhood, gastrointestinal infections, such as enteritis, typhoid fever and dysentery, peritonitis, including mesenteric adenitis and tabes mesenterica; liver diseases, such as catarrhal jaundice; enlargement of the spleen and liver; enlargement of the urinary bladder; and the rare, but little understood, instances of gastric and duodenal ulcer in children, were mentioned.

In discussing the "Surgical Conditions Causing Abdominal Pain," Dr. Gabriel Rubin spoke first of acute appendicitis, in which pain is usually the first symptom and is generalized in 80 per cent and localized in 20 per cent of the instances. Peritonitis that is secondary to some abdominal condition is characterized by severe pain, marked spasm and vomiting; primary peritonitis, such as that caused by the pneumococcus, has very similar signs and is best treated by operation after a delay to allow the infection to become localized. Three obstructive lesions are the chief causes of abdominal pain in infancy, which is signalized by the baby drawing up his legs and failing to be calmed by defecation or by attempts to make him comfortable. With its peak of incidence in the third week of life, pyloric stenosis is satisfactorily treated by pylorotomy. Incarcerated inguinal hernia demands immediate operation. Intussusception begins with a sudden onset of pain, followed by collapse, pallor and listlessness, and the passage of blood by rectum; early operation is indicated. Among the rarer causes of pain, inflammation of Meckel's diverticulum, volvulus and twisted ovarian cyst may be noted. In the case of a ruptured hollow or solid viscus, pain may not appear until after shock is evident.

The subject taken by Dr. Jacob Wallace was "Functional or Neurogenic Pain," with particular attention to the functional disorders of the gastrointestinal tract. The so-called irritable colon, which is often made more irritable by the unwise use of roughage and laxatives, may give rise to either diarrhea or constipation or to both. The racial incidence of this condition is particularly high among Jewish children. The usual complaint is abdominal pain, either in one quadrant or generalized, that typically follows the course of the colon, is more severe just before defecation and is often initiated by food. Nausea is a common symptom, but vomiting is a rare one; anorexia is most marked early in the morning. The diagnosis may be made after organic disease has been ruled out by sigmoidoscopy and x-ray and is established with more assurance in children than it is in adults, because of the relative rarity of malignant processes at this age. Since it it is possible to confuse this condition with appendicitis, careful inquiry should be made concerning the dietary and bowel habits and the emotional state of the patient. The treatment consists in removing all roughage and condiments, exhibiting antispasmodics and establishing a regular daily program conducive to emotional stability. Cyclic vomiting is a condition beginning after infancy and stopping at 12 years, which is characterized by a great deal of

vomiting associated with acetonuria. Appropriate therapeutic measures are bed rest, forcing fluids and elimination of solid food. Operation, in the belief that the patient has appendicitis, may prove fatal. Dilatation of the colon or Hirschsprung's disease, causes meteorism, obstipation and a palpable colon.

Dr. Morris Ingall described a large group of diseases under the title of "Nonacute Conditions with Atypical Abdominal Signs." He emphasized the importance of doing as many laboratory tests as are necessary and of taking a careful history of the patient before rushing him into an operation. Children with pica often ingest plaster, hair, wool, paste, paper, paint, and so forth, and may develop trichobezoar or chronic lead poisoning with encephalopathy or colic. Diabetic acidosis produces nausea, vomiting, abdominal pain, hyperpnea and slight fever; an acute surgical condition of the abdomen may coexist. Pinworms are the only common intestinal parasite found in this region. Gastrointestinal allergy may result in pain after the ingestion of an offending antigen, while abdominal pain with a rising fever is a common early symptom of serum sickness. Rheumatic fever may cause a dull ache, with slight tenderness but no rigidity. Biliary colic is very rare, but acute catarrhal jaundice may begin with nausea, vomiting and pain in the abdomen. So-called abdominal migraine and certain brain diseases causing an abdominal condition consistent with that of appendicitis may be confusing. Among other infrequent causes of abdominal pain, Henoch's purpura, visceroptosis, Dietl's crisis, Pott's disease, trichinosis, herpes zoster, periarteritis nodosa and pertussis may be mentioned.

The final paper was presented by Dr. Solomon Rubin on the "Approach to the Child." Since a great variety of temperaments are inevitably to be encountered, all must be understood to be handled successfully. The treatment of the parent often requires considerably more skill than the treatment of the sick child. In analyzing the psychologic reactions of the child, one must consider the following factors: inheritance, which may be judged from the family history; changes produced by infection and disease working on a comparatively unstable nervous system; anatomic, physiologic and psychologic differences between children and adults; and, difficulties of the neuropathic child in making adjustments in the home, school and doctor's office. The importance of the illness may not be measured by the intensity of the subjective symptoms, which are often very much exaggerated. Because children are not able to localize pain accurately, the demonstration of tenderness by physical examination is much more reliable as a guide to the region where disease is present.

In concluding the symposium, Dr. Eli Friedman emphasized the fact that, although cases of abdominal pain of a medical nature outnumber those of a surgical nature in a ratio of approximately 85 to 15, it is imperative to recognize the surgical cases early in order to reduce the mortality from such conditions.

SPECIAL LECTURES IN ENDOCRINOLOGY

"The Female Sex Hormones in Pregnancy" was the subject of a lecture delivered by Dr. George van S. Smith at the Harvard Medical School, March 4.

In studying the female sex hormones in pregnant women, the problem of quantitative analysis is less difficult because such large amounts of estrin and A.P.L. (anterior pituitary-like hormone of placental origin) are present that urine and serum may both be assayed with a fair degree of accuracy. The introduction of acid hydrolysis in the quantitation of urinary estrin has resulted in much higher values than have been obtained formerly, 50,000 to 100,000 rat units being present in 24-hour volumes of urine after the eighth month.

After describing in some detail the methods of extraction and assay for these two hormones, Dr. Smith emphasized the reciprocal relation between them in the body. High levels of A.P.L. are associated with relatively low levels of estrin and vice versa, except during the first 3 weeks following the implantation of the ovum, when the amounts of these hormones increase simultaneously in the urine.

He then demonstrated curves of estrin excretion during the month of conception and the first 3 months of the ensuing pregnancy and also curves of A.P.L. excretion from the time of its first appearance after conception. The curves of estrin excretion during the month of conception are entirely similar to those previously presented as typical of normal ovulatory cycles until the twenty-fifth day, after which the estrin increases rapidly instead of dropping off as it does before normal menstruation. This second rise in estrin probably marks the time of implantation of the fertilized ovum. In one instance, such a rise was followed by a subsequent drop and bleeding 8 days after menstruation was due. This was interpreted as a very early abortion.

A.P.L., which gives the positive tests for pregnancy, is demonstrable soon after the missed catamenia and increases rapidly, reaching a peak at approximately the second month, at which time the estrin levels off or actually decreases, apparently due to the high A.P.L.

Curves for serum A.P.L. and estrin from about the second month to the end of normal pregnancy were shown. By the fourth month, the A.P.L. strikes a fairly low level, which continues to term, and the estrin begins to rise, reaching its peak at or near term. The drop in A.P.L. and the rise in estrin coincide with the cessation of early nausea and vomiting. The shapes of the urinary curves for these hormones are so similar to the serum curves that the urinary values may be accepted as reflecting the serum levels.

The regression of the corpus luteum of gestation does not mean a cessation of the supply of progestin, for progestin is found in significant amounts in placentas in the last half of pregnancy. Since placentas from normal, full-term deliveries are also found to contain progestin, the precipitation of labor cannot be explained by the complete absence of this

hormone in the presence of large amounts of estrin. Moreover, serum estrin has been found to be no higher at delivery than before labor started. It is possible, however, that a change in the quantitative balance of these two hormones is involved in the initiation of labor. Although accurate, quantitative, progestin determinations are still impossible, assays of 19 placentas from 6 months to term from both normal and toxemic cases showed no differences in the content of progestin sufficiently pronounced to be correlated with the clinical picture.

A study of A.P.L. and estrin in the serum, urine and placenta of patients with toxemias of late pregnancy revealed a marked excess of A.P.L. and rather low levels of estrin as being typical of this disease. This excessive A.P.L. was demonstrable in the serum 6 to 8 weeks before the development of clinical symptoms. A striking lag in the excretion of A.P.L. was seen in these cases of toxemia, this observation being the only instance in which urinary and serum values failed to run parallel and apparently resulting from a change in kidney threshold for A.P.L. excretion. The increase in circulating A.P.L. would account for the comparatively low levels of estrin in toxemic patients. The hormonal and pathologic analogy between the toxemic situation and that which occurs during the premenstrual phase in nonpregnant ovulatory cycles was pointed out as suggestive of the possible etiology of and therapeutic approach to late pregnancy toxemia.

THE MASSACHUSETTS SOCIETY FOR SOCIAL HYGIENE

The Massachusetts Society for Social Hygiene held its annual meeting April 13 in Boston. The guest speakers were Dr. Ray Lyman Wilbur, president of Stanford University and of the American Social Hygiene Association, and Dr. John R. Heller, Jr., assistant surgeon of the United States Public Health Service.

The economic importance of syphilis, as well as its ravages on the health of the people, was emphasized by the speakers. This meeting was a valuable contribution to the program now under way to instruct the people of this country about the necessity of action in dealing with this menace to life and health.

The officers elected for the ensuing year are: Dr. George Gilbert Smith, president; Mrs. Harry C. Solomon and Miss Elizabeth Ross, R.N., vice-president and secretary, respectively; and William Wadsworth, treasurer.

The society is to be congratulated in securing for its president another prominent and enthusiastic worker in this field to carry on the work inaugurated by Dr. Crabtree.

SOUTHEASTERN MASSACHUSETTS ASSOCIATION OF BOARDS OF HEALTH

For its spring meeting the Southeastern Massachusetts Association of Boards of Health chose the Christian Church in Smith Mills, North Dartmouth, with some forty members and invited guests in attendance. In the absence of the president, the meeting was called to order by Mr. Joseph E. Christie, vice-president and a member of the Dartmouth Board of Health. The principal speaker was Dr. C. W. Milliken, school physician of New Bedford. He discussed the proposed legislation which would effect the transfer of the supervision of health in public schools from the local school department to the board of health. His position was in opposition to the proposed change; he urged that the existing conditions are more advantageous to the towns. He said that the school committees in general are able to secure excellent results today and are working in close cooperation with the health departments, and added that, in the smaller towns, especially those in which there is no all-time health officer, there would be a distinct disadvantage compared with present conditions. Dr. H. H. Mandell, of New Bedford, spoke in support of Dr. Milliken, delving into past history to the time when it was a health physician who inspected the schools. "It was a good thing for the schools," he said, "when this inspection was taken from the health department and given to the school committee." Dr. R. P. MacKnight, of New Bedford, state district health officer, was a third speaker.

BOSTON UNIVERSITY SCHOOL OF MEDICINE ALUMNI

The annual dinner meeting of the alumni of the Boston University School of Medicine was held at the Hotel Kenmore on April 29. Dr. Sanford B. Hooker acted as toastmaster, and the speakers included Dr. Alexander S. Begg, dean of the Boston University School of Medicine, Dr. LeRoy M. S. Miner, dean of the Harvard Dental School and a member of the faculty of the Boston University School of Medicine, Dr. Daniel L. Marsh, president of the University, Dr. Wesley T. Lee and Anthony Palma, president of the graduating class.

Plans for launching a campaign to raise funds for a new building, equipped with modern laboratories, to replace the sixty-five year old building on East Concord Street were discussed. Tentative plans have been drawn up of a red brick structure, extremely simple in design, with stone facings. The plans for this building in no way affect the proposed plans to erect a quadrangle of buildings for the University on its Bay State Road campus site. The exact amount of the funds to be raised will be determined at the next meeting of the trustees. Some funds, mostly small gifts from alumni, are already available.

MASSACHUSETTS SOCIETY OF EXAMINING PHYSICIANS

The annual dinner meeting of the Massachusetts Society of Examining Physicians was held at the Copley-Plaza Hotel on May 5 and was dedicated to the memory of the late Dr. Francis D. Donoghue, who founded the society in 1907.

The speakers included Joseph Parks, chairman of the Industrial Accident Board, Judge Frank J. Donahue, Judge Nelson Brown, Dr. Frederic J. Cotton, former surgeon-in-chief of the Boston City Hospital, Edward Clark, former member of the Industrial Accident Board, and Dr. William E. Browne, clinical professor of surgery at Tufts College Medical School.

The following officers were elected: Dr. Francis R. Mahony, of Lowell, president; Dr. William J. Brickley, of Boston, vice-president; Dr. William P. Coues, of Brookline, secretary; Dr. Robert C. Gwin, of Brookline, treasurer.

LYNN CANCER CLINIC

The Lynn Cancer Committee held a teaching clinic, conducted by Dr. Frank H. Lahey, of Boston, at the Lynn Hospital, Thursday, April 29, as part of the celebration of the tenth anniversary of the Lynn Cancer Clinic.

On the same date, a public luncheon was held, at which a report of the activities of the Lynn Cancer Clinic during the past ten years was made. Speakers on this occasion were Dr. Henry Chadwick, commissioner of public health, and Dr. Lahey.

THE AMERICAN ASSOCIATION OF THE HISTORY OF MEDICINE

At the annual meeting of the American Association of the History of Medicine held at Atlantic City, May 3, the following officers were elected: president, Dr. Walter R. Steiner, of Hartford, Conn.; vice-president, Dr. E. R. Long, of Philadelphia; secretary-treasurer, Dr. E. J. G. Beardsley, of Philadelphia; council, Joseph L. Miller, of Chicago; Carl V. Weller, of Ann Arbor; Henry E. Sigerist, of Baltimore; William S. Middleton, of Madison, Wis., and Jabez Elliott, of Toronto.

ANNUAL BANQUET AND BALL OF MIDDLESEX COLLEGE

Dr. Stephen Rushmore, chairman of the Approving Authority on Medical Education and Secretary of the Board of Registration in Medicine, was the guest of honor and principal speaker at the sixteenth annual banquet and ball of Middlesex College conducted by the Alumni Association of the School of Medicine on April 15 in the Imperial Ballroom of the Hotel Statler. Dr. Rushmore addressed the gathering of 700 guests on the subject of "Medical Education—Now and Then."

Describing the changes in medical education over a period of years, Dr. Rushmore pointed out that there has been a gradual spreading and broadening of standards which were in existence forty years ago in at least one institution, Johns Hopkins University School of Medicine, of which he is an alumnus. The requirements for admission then established at that institution, as part of a unique experiment in education, were graduation from college, a reading knowledge of French and German, and one year each of chemistry, physics and biology, and the present requirements have now been increased by the addition of only about one more year of chemistry than was required forty years ago.

The great progress that has been made generally in medical education in these comparatively few years was ascribed by Dr. Rushmore to two principal causes: the transforming influence of the university spirit, since most medical schools today are parts of universities, and the driving power of discovery in the fundamental sciences underlying medicine, particularly in the x-ray field, in chemistry and in the other premedical sciences.

"The burden of medical education must be carried today, not by the person who teaches once in a while, but by the person proficient in science by long training who makes medical education his major interest. Today is the day of the expert. Others may participate, but the burden of medical education must be borne by these experts."

Discussing the problem of how many medical schools there should be in this country, Dr. Rushmore said, "When you ask physicians, they will say there is too much competition; if you ask candidates, they will tell you there are too few schools. Today out of 12,000 annual candidates for admission, about 7,000 are crowded into first-year classes of our medical schools, and the remaining 5,000 are excluded. On that basis ten more schools might be needed to remove the pressure temporarily, but medical education must be a permanent affair. The number of medical schools should not be determined arbitrarily by any body or bureau, for no one knows how many physicians there should be for the 125,000,000 people in this country."

In discussing the present needs of medical education, Dr. Rushmore concluded, "We need money, buildings, books and libraries for medical schools, but more than anything else we need ideas—new and better ways of doing new and better things. It is not sufficient to have ideas. We must master them and know how to apply them, so that they may become effective realities in this actual world of sickness and suffering in which we live and which we are striving in some small measure to improve."

Horace T. Cahill, speaker of the House of Representatives, also gave an address. He warned the medical profession that its independence might be challenged by crackpot schemes of socialization and that no longer could it maintain a provincial attitude. For their own protection, he said, members of the medical profession must interest themselves in government, in politics, even in vote-getting, if they are to preserve their independence. Speaker Cahill declared that physicians could make a genuine contribution to government as well as to the safe-being of their own profession, if they would aspire to elective offices higher than the traditional positions on local school committees.

Other speakers and guests at the banquet included Dr. Frank L. Whipple, dean of Middlesex College; Dr. Howard Charles Gale, Dr. Horatio S. Card, Rev. Hugh Wallace Smith and John M. Rus-

sell, trustees of Middlesex College; Mayor John D. Lynch, of Cambridge; Judge Patrick J. Duane, of Waltham; and a delegation from the Massachusetts Legislature.

NOTICES

REMOVAL

ALPHA R. SAWYER, M.D., announces the removal of his office to 416 Marlboro Street, Boston.

SPECIAL MEETING OF THE ACADEMY OF PHYSICAL MEDICINE

A special business meeting of the Academy of Physical Medicine will be held at 5:30 o'clock on Wednesday, May 19, at 144 Commonwealth Avenue, Boston.

HERMAN A. OSGOOD, M.D.,
Secretary-Treasurer.

POSTPONEMENT OF MEETING OF THE NEW ENGLAND PHYSICAL THERAPY SOCIETY

The annual meeting of the New England Physical Therapy Society will be postponed to the evening of May 26.

The program will appear next week.

WILLIAM D. MCFEE, M.D., *Secretary.*

BOSTON CITY HOSPITAL

A clinical meeting of the staff of the Boston City Hospital will be held in the Thorndike Amphitheater, Boston City Hospital, on Saturday, May 22, at 10:00 a. m.

PROGRAM

1. The Study of Alcoholism at the Boston City Hospital. Dr. Merrill Moore.
2. A New Series of Anticonvulsants, Tested on Animals. Dr. H. Houston Merritt and Dr. Tracy J. Putnam.
3. Epilepsy—A Paroxysmal Cerebral Dysrhythmia. Dr. William G. Lennox, Dr. F. A. Gibbs and E. H. Gibbs.
4. Migraine and Its Treatment. Dr. Theodore J. C. von Storch.
5. The Results of Treatment of Athetosis and Dystonia by Section of the Extrapyramidal Tracts. Dr. Tracy J. Putnam.

Committee on Hospital Clinics.

TRUDEAU SOCIETY

A meeting of the Trudeau Society will be held at the Belmont Hospital, Worcester, on Wednesday, May 19, at 4 p. m. The following program has been arranged:

1. Address of Welcome. Dr. A. C. Getchell.
2. Results of Fifty-Six Phrenic Operations. Dr. N. O. Furnas.
3. Major Surgery in a Small Tuberculosis Hospital —Presentation of Cases Operated upon in This Hospital. Dr. M. Thoroman.
4. Presentation of Nonpulmonary Tuberculosis Cases with X-Rays and Pathologic Specimens. Dr. R. H. Smith and Dr. R. Goodale.
5. Presentation of More Than Thirty Cases of Empyema Treated with Gomenol. Dr. R. H. Baker.

NEW ENGLAND WOMEN'S MEDICAL SOCIETY

The spring meeting of the New England Women's Medical Society will be held at the Hotel Puritan, May 20, at 7 p. m. The principal speaker will be Dr. Miriam Van Waters of the Massachusetts Reformatory for Women. The charge for dinner will be $1.50 per plate.

MARY I. TOMPKINS, M.D., *Secretary.*

NORTHERN WORCESTER COUNTY PUBLIC HEALTH ASSOCIATION

Dr. John B. Hawes, 2d, president of the Boston Tuberculosis Association, will speak at the annual meeting of the Northern Worcester County Public Health Association at the Hotel Raymond, Fitchburg, at 12.30 p. m. on May 19.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, MAY 17

Monday, May 17—

*8 p. m. Lecture on alcoholism, Dr. Robert Fleming, in the amphitheater of the Mallory Institute of Pathology, 784 Massachusetts Avenue, Boston.

Tuesday, May 18—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Some Thoughts on the Functions and Possible Pathological Significance of the Renal Nerves. Dr. G. Philip Grabfield.

9:30 a. m. Massachusetts General Hospital. Thoracic Clinic. Ether Dome.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

Wednesday, May 19—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital case presentation. Dr. S. J. Thannhauser.

†12 m. Clinicopathological conference. Children's Hospital amphitheater.

2 p. m. Massachusetts General Hospital. Psychiatric clinic. Out patient department.

4 p. m. - 5 p. m. Surgical pathological conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

5:30 p. m. Special meeting of the Academy of Physical Medicine. 144 Commonwealth Avenue, Boston.

Thursday, May 20—

8 a. m. Massachusetts General Hospital. Circulatory clinic rounds.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Cardiac Irregularities in Bacterial Endocarditis. Dr. Maurice S. Segal.

11 a. m. Massachusetts General Hospital. Medical grand rounds. Ether Dome.

12 m. Massachusetts General Hospital. Clinicopathological conference.

7 p. m. New England Women's Medical Society. Hotel Puritan, Boston.

Friday, May 21—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. The Origin and Nature of Complications of Scarlet Fever. Dr. Conrad Wesselhoeft.

12 m. Massachusetts General Hospital. Urological conference. Out patient department.

Saturday, May 22—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital case presentation. Dr. S. J. Thannhauser.

10 a. m. Boston City Hospital, Clinical Meeting of the Staff. Thorndike Amphitheater.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

May 13—Pentuckét Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

May 14—Cutter Lecture on Preventive Medicine, 5 p. m. Harvard Medical School, Building E.

May 18—South End Medical Club at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

May 19—Northern Worcester County Public Health Association. Page 865.

May 19—The Trudeau Society. Page 865.

May 19—Special meeting of the Academy of Physical Medicine. Page 865.

May 19, 20—The Connecticut State Medical Society, Bridgeport.

May 20—New England Women's Medical Society. Page 865.

May 22—Boston City Hospital, Clinical Meeting of the Staff. Page 865.

May 26—Postponement of the meeting of the New England Physical Therapy Society. Page 865.

June 1, 2 and 3—Annual meeting, Massachusetts Medical Society, Hotel Bradford, Boston.

June 7—American Medical Golfers Play in Atlantic City. Page 582, issue of April 1.

June 7 and 8—American Heart Association. Page 769, issue of April 29.

June 7-11—American Medical Association. Atlantic City. Page 709, issue of April 22.

June 8—The American Neisserian Medical Society. Page 489, issue of March 18.

June 8 and 9—American Board of Obstetrics and Gynecology. Page 78, issue of January 14.

June 11-18—Catholic Hospital Association. Page 488, issue of March 18.

July 6-11—Fifth International Congress of Hospitals. Page 37, issue of January 7.

September 13-17—Fifth International Congress of Radiology. Page 709, issue of April 22.

October 5-8—American Public Health Association meeting. New York City.

October 9—American Board of Ophthalmology, Chicago. Page 722, issue of April 22.

October 19, 20, 21—Academy of Physical Medicine. Page 723, issue of April 22.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETY

PLYMOUTH DISTRICT MEDICAL SOCIETY

May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

BOOKS RECEIVED FOR REVIEW

Surgical Pathology of the Thyroid Gland. Hertzler's Monographs on Surgical Pathology. Arthur E. Hertzler. 298 pp. Philadelphia, Montreal and London: J. B. Lippincott Company, 1936.

Cataract. Its Preventive and Medical Treatment for Specialists, General Practitioners and Students. A. Edward Davis. 161 pp. Philadelphia: F. A. Davis Company, 1937. $3.00.

Diabetes. A Modern Manual. Anthony M. Sindoni, Jr. 240 pp. New York and London: Whittlesey House, McGraw-Hill Book Company, Inc., 1937. $2.00.

Fruits of Philosophy or the Private Companion of Adult People. Charles Knowlton. 107 pp. Mount Vernon: Peter Pauper Press, 1937.

The Ocular Fundus in Diagnosis and Treatment. Donald T. Atkinson. 142 pp. Philadelphia: Lea & Febiger, 1937. $10.00.

Sexual Power. Chester Tilton Stone. 172 pp. New York and London: D. Appleton-Century Company, 1937. $1.50.

Trauma and Disease. Edited by Leopold Brahdy and Samuel Kahn. 613 pp. Philadelphia: Lea & Febiger, 1937. $7.50.

Biological Time. P. Lecomte Du Noüy. 180 pp. New York: The Macmillan Company, 1937. $2.00.

Les Gastropathies des Syphilitiques. Carlos Bonorino Udaondo. 214 pp. Paris: Masson et Cie, 1936. 32 fr.

The Thyroid and Its Diseases. J. H. Means, 602 pp. Philadelphia, Montreal, London: J. B. Lippincott Company, 1937.

Traité de Biocolloidologie. Tome IV. État Colloïdal et Biologie. W. Kopaczewski. 924 pp. Paris: Gauthier-Villars, 1936.

Physiology in Health and Disease. Carl J. Wiggers. 1124 pp. Second Edition, Thoroughly Revised. Philadelphia: Lea & Febiger, 1937. $9.00.

Estudos Cirurgicos. 2. a serie. Eurico Branco Ribeiro. 245 pp. Sao Paulo, Brasil: Sociedade Editora Medica Limitada, 1936.

BOOK REVIEWS

Endocrinology in Modern Practice. William Wolf. 1018 pp. Philadelphia and London: W. B. Saunders Company. $10.00.

This volume was written to provide the practitioner with the usable facts of clinical endocrinology. The author further hopes it will serve to make the specialist endocrinology-minded and toward this end has devoted considerable space to the endocrine aspects of the more commonly practiced specialties.

In undertaking this tremendous task, the author has first presented a brief description of anatomic, physiologic and biologic characteristics of each of the endocrine glands. Then follow descriptions of the various syndromes which are thought to represent primary dysfunctions of these glands. The various pluriglandular syndromes are described under the section pertaining to the gland which is thought to be primarily responsible. The therapeutic measures indicated in each syndrome described are briefly outlined. An attempt is made to discuss the effects of other diseases upon the function of the various endocrine glands. Chapters pertaining to the interpretation of laboratory findings, choice of diagnostic procedures, methods for performing some of the more commonly employed laboratory procedures, and the manner of proceeding with the working up of a supposed endocrine case, are included. In order to facilitate matters for the busy practitioner, a chapter is devoted to symptom diag-

nosis. Here each symptom and the endocrine disorder in which it may occur are listed. Similar tabular data concerning the various endocrine products and their dosage are presented.

One wonders if much of the matter covered is not in too great a state of flux to warrant presenting it in such detailed form to the general practitioner. The volume contains much which can be of no practical value to the general practitioner. Therapy prescribed in many instances does not seem to be based on accurate scientific study. It would seem better to present the subject of clinical endocrinology to the practitioner in as brief a form as possible, devoid of as many controversial clinical facts as possible, thereby giving him a much firmer basic understanding upon which to build. The volume contains no bibliography and therefore is not of so much interest and value to the endocrinologist.

A Manual of Practical Obstetrics. O'Donel Browne. 363 pp. Baltimore: William Wood & Company. $6.50.

The just fame of the Rotunda Hospital in Dublin warrants high expectation from this well-written manual by the Assistant Master. So different, however, are the viewpoints in obstetrics on this side of the Atlantic, it cannot be considered as indispensable to either the student or general practitioner here. Our effort in the United States to improve maternity results are directed to extension of hospital facilities and methods to all but quite normal cases. Many methods described in this book, such as the use of a vaginal plug for placenta praevia, are those better adapted to home operating, which we have decided is outmoded. However, the conscientious practitioners in inaccessible rural districts, as yet untouched by good highways, who cannot promptly remove the abnormal patient to a good hospital, may find here much useful instruction. One wonders, though, whether the directions for even the simplest operations can be followed by a doctor so inexperienced as to need the picture of a needle-holder.

The kind of midwifery that the author has in mind may be illustrated by these sentences from page 61. "The real success in the first stage is in the hands of the nurse. It is only fair to tell her exactly what she is to do, if she is not already fully aware of your wishes. She should be able to recognize the change from first to second stage labour and judge the time to send for medical assistance for delivery."

Many other directions throughout the book recall the divergence in cisatlantic ideas. On page 72, one is warned never to use pituitrin after the first stage of labor. Many here believe it is safest after the cervix is fully dilated. On page 104, pituitrin is said to be bad for primary inertia, and what is called "masterly inactivity" is urged. On page 74, nitrous oxide and oxygen are said to be "ideal for toxic cases." Many here believe that hypertension is increased, as well as the danger of capillary rupture by the almost inevitable cyanosis, if complete anesthesia is induced with nitrous oxide. Basal anesthetics are discountenanced, and paraldehyde and the barbiturates, widely and successfully used in this country, are not even mentioned by name.

The description of normal labor, of abnormalities and of the pathology involved is good; but the instructions for treatment are so at variance with accepted procedures in this country as to be quite confusing to the uncertain novice. On page 147, for the treatment of contraction ring, it is suggested, "if delivery is indicated," that forceps should be tried at first. "If this step fails to dilate the ring, bring down a leg and apply steady prolonged traction." If version is found impossible because of tightness of the spasm, cesarean section is then advised. In America we believe that abdominal section should never be considered an escape method; following unsuccessful forceps or version it is exceedingly dangerous and contributes generously to maternal mortality.

The drawings are many and good, especially those of pelvic pathology, although picturing the right hand as the vaginal one in manual examination is again somewhat confusing to American trained obstetricians. There is an interesting chapter on radiology and another on blood-grouping, although the great value of transfusion as a prophylactic procedure in bleeding cases and in the treatment of shock is not emphasized.

The book is well bound in cloth, the paper is heavy and the printing clear. There is an excellent, detailed index.

Bulletin of the Neurological Institute of New York. Edited by Charles A. Elsberg. Volume 5, August, 1936. 544 pp. Baltimore: Waverly Press, Inc.

This volume is the Elsberg Anniversary Number and marks his sixty-fifth birthday.

Charles A. Elsberg has done much for the advancement of neurosurgery and, in particular, the surgery of the spinal cord. It is fitting that his sixty-fifth birthday and his retirement as the active leader of the surgical service of the Neurological Institute should be recognized by his friends and pupils.

The volume consists of some thirty-three papers on neurologic and neurosurgical subjects. Many of these papers are of real value, and almost all of them are interesting. The great variety of subjects discussed in these papers shows the energy of the staff of the Institute and the wealth of material at its disposal. It is not the desire or purpose of the reviewer to select any article or articles in a Festschrift of this sort for especial praise. All were written—whether by Dr. Elsberg's oldest associate or by his youngest pupil—to do him honor. It is sufficient to say that the average value of these contributions is high and, therefore, the whole

book is well worth reading. The printing and the illustrations are excellent, but it is a pity that the proofreading was not done more carefully. A brief "Foreword" by Frederick Tilney summarizes Elsberg's life and estimates his value as a neurosurgeon. A portrait of Elsberg and a useful index complete the volume.

An Index of Treatment. By various writers. Edited by Robert Hutchison. Eleventh Edition. 1020 pp. Baltimore: William Wood & Company. $12.00.

It is a notable attainment for this publication to reach its eleventh edition since its first appearance in 1907 — this itself proves merit. It is an encyclopedic compilation written by many English physicians of high standing aiming to provide the practitioner with a complete guide to treatment in a compact and convenient form. It is well written. The topics are classified alphabetically, and the subject matter is treated authoritatively and concisely. There is always emphasis on common sense and practicability, and principles are well stressed. There is a tendency, however, to polypharmacy, and details bespeak, at times, therapeutic customs not familiar to us. A more important criticism, however, is its fixed bulkiness and high cost for a reference work on a subject which is steadily changing. A loose-leaf arrangement ought to make this otherwise excellent book more practical and lasting to the general practitioner.

A Textbook of Obstetrics. Edward A. Schumann. 780 pp. Philadelphia and London: W. B. Saunders Company. $6.50.

Rarely is a reviewer justified in giving such unqualified commendation as this book frankly merits. It is a joy to find a textbook so perfectly adapted to its purpose. Everything pertinent to the science and the art of obstetrics is included, and fine discrimination is shown in awarding plates and space to the plenitude of subjects discussed. If perhaps a few more pages are given to the discussion of ectopic pregnancy than the subject would seem to merit, the reader will not cavil, because of the excellence of the subject matter and the illustrations. The major subjects of abortion, placenta praevia, toxemia and sepsis are ably presented, and the student will derive much profit from the author's long-standing interest in, and experience with, the mechanics of labor and delivery.

Here and there, of course, the justly envious reader will joyfully pounce upon such trivialities as the failure to mention high blood estrogen as the stimulating agent in cancer complicating pregnancy; or that the role of vitamin B is not mentioned in the discussion of polyneuritis; or that germ plasm deficiencies are not given their share of blame for habitual abortion. Such details do not detract. Indeed, were the literary style not so fine and the fulsomeness of suggestion not so consistent and extensive, the reader would not himself be kept up to the intellectual pitch which makes him aware of the rare omission.

The make-up of the text is exceedingly serviceable. Under the chapter heading, the discussion is separated into subchapters, main paragraphs and subparagraphs, all clearly demarked by various type. This will prove of great assistance to the undergraduate student, for it helps him to think logically and to remember. The book is not so large and heavy as most comprehensive textbooks, being but slightly larger than the conventional novel. The paper is pleasantly smooth and shiny and the type, perfect. There is a useful, sensibly concise bibliography, with no attempt at display, and an adequate index. The book is well worth its cost. We congratulate Dr. Schumann, who serves obstetrics well by this admirable treatise; and we commend his publishers, W. B. Saunders Company, for their good bookmaking.

A Textbook of Surgery. John Homans. 1267 pp. Fourth Edition. Springfield and Baltimore: Charles C Thomas. $8.00.

This textbook of surgery, written by Homans with the aid of various members of the Surgical Department of the Harvard Medical School, has already gone through three editions since it was first published in February, 1931. In this, the fourth edition, the book has been revised and a chapter on Amputations and Plastics has been added.

One is accustomed to think of a textbook of surgery as a dry compilation of essential facts, valuable for reference but hardly an ideal companion for an evening by the fire. Great was the surprise of this reviewer when he found himself led on, page after page, by the delightful way in which Homans has presented his material. Every chapter opens with a piquant historical sketch of the early development of the branch of surgery to be described. "That astonishing old genius, Hippocrates," is a masterful summation. The author does not confine these touches to his introductions. His conclusion to the description of the formation of a bedsore, "thus a very unpleasant sort of ulcer is established," is an example of the original kind of humor which lightens many a page.

For his ability to present the fundamental facts connected with every sort of surgical problem the author deserves the highest praise. The treatment of these conditions must necessarily, in a book like this, be simply an outline, but it is remarkable how successfully Homans has condensed this phase of the subject.

The inclusion of the specialties in a book on surgery is unusual, but Homans has excellent chapters, for which he gives credit to his allies, on the Eye, Ear, Nose and Throat, and Neurologic Surgery. Undoubtedly a specialist in each of these fields might

find minor omissions and inadequate representation of problems which to him seem highly important, but the medical student covering the entire field of surgery cannot go wrong if he remembers the things Homans emphasizes.

The book is excellent, and well deserves the successful reception that has been given it.

Preoperative and Postoperative Treatment. Robert L. Mason. 495 pp. Philadelphia and London: W. B. Saunders Company, 1937. $6.00.

The author, with the help of eleven collaborators, undertakes to present a detailed consideration of the preparation and aftercare of surgical patients. In general, the plan and presentation are excellent, and the book should be of great value to the general surgical practitioner. Adequate illustrations supplement the text, and there are an appendix of normal chemical standards and a good working index. Bibliographies at the ends of most chapters refer to the chief studies in the recent literature as well as to some of the older, classical studies.

A valuable feature is the detailed presentation of diets suitable for various problems. These are quoted from the "Diet Manual" of the Massachusetts General Hospital, where clinical trial has justified their usefulness.

In preparation of such a book, the author must have more than the ordinary indecisions as to what to include and what to reject. Chapters dealing with Shock and Water Balance are obviously necessary, although their applicability extends to wider fields than the intention of this book. An excellent chapter on Superficial Burns, by Aldrich, is less pertinent, but will undoubtedly be of interest and value to readers.

No attempt is made to discuss the field of neurosurgery, nor is there any discussion of thoracic surgery save for a chapter on Acute Empyema and Subphrenic Abscess by Strieder.

Additional chapters would be desirable on special considerations affecting surgery in children, surgery in vascular diseases, and plastic surgery. Skin preparation is covered in one brief paragraph in the chapter on General Preparation, and another in the chapter on Orthopedic Surgery. However, there is no discussion of skin preparations in emergency surgery or of the use of skin antiseptics on the operating table. Some reference to the role of physiotherapy in postoperative care of traumatic and orthopedic cases would be useful. Munro's method of tidal drainage of the bladder is twice alluded to, but an illustration, an explanation of the apparatus, and a reference in the index would be helpful.

Part I of the book deals with general considerations, and the titles of chapters, such as Operative Risk, Heart Disease, Diabetes, Anesthesia, Transfusion, Pulmonary Complications, Ileus, Wound Disruption, Urinary Complications, and the like, sufficiently indicate its scope. Part II deals with regional surgical problems — Otolaryngology, Hyperthyroidism, Biliary Tract, Hernia, Gynecology, Urology, Traumatic Surgery, and so forth.

Many of the chapters dealing with special fields and problems have been contributed by collaborators who, in general, are well qualified for their tasks. As in all such books written with the help of collaborators, however, the chapters are unequal and uneven as to merit, length, character and emphasis; and a certain amount of repetition, duplication and disagreement seems to be inevitable.

Light Therapy. Frank Hammond Krusen. Second Edition, Revised and Enlarged. 238 pp. New York: Paul B. Hoeber, Inc., 1937. $3.50.

This book contains a wealth of valuable up-to-date information on three portions of the electromagnetic spectrum—ultraviolet, visible and infrared—and is written by one well qualified to do so. It contains chapters on the physics, sources and physiology of these rays, suggests the relative value of most of the more common artificial sources producing these radiations, and gives the technic for applying them.

More than 70 pages are devoted to various diseases for which this type of treatment has been advocated, emphasizing the indications and contraindications for such use. Throughout the book the author has quoted freely from authorities in this work, interspersing these quotations with notes of explanation and suggestion. By this method he permits the reader to be the judge of the efficiency of the various sources of light and heat when dealing with certain diseases.

The author has blazed the trail for a better understanding of radiation therapy but emphasizes our present limited knowledge in this field. Such a presentation forces the reader to think for himself and is stimulating for research.

A most disappointing feature, however, is the prominence given to the many diseases appearing at the beginning of nine of the chapters on ultraviolet radiation. These diseases, two hundred and fifty of them, are labeled "in which ultraviolet light has been said to be of value," because someone published an article following the successful outcome of a case in which ultraviolet light was used. This detracts a great deal from the value of the book, as a large number of these diseases could be helped by ultraviolet light only through some very circuitous route and a vivid imagination. The casual reader might be easily misled into thinking otherwise.

The author is highly capable of presenting the reader with an authoritative list of the few pathologic conditions for which ultraviolet radiation is of value, and then, if he should choose, of adding a list of the few other conditions about which there is still some doubt regarding the efficacy of this treatment. Some such arrangement would greatly increase the value of the book and, in addition, would render it much more practical. The author has apparently included these with the name of the writer only to make his book impartial and not be-

cause he always agrees. Nevertheless, this book is a distinct asset in the field of physical medicine and is worthy of a place in the working library of all those physicians who are attempting to use physical medicine seriously.

Brookings. A Biography. Hermann Hagedorn. 334 pp. New York: The Macmillan Company. $3.50.

The reason for reviewing this charming biography in a medical journal is the close relation between Brookings's career and the renascence of the medical school of Washington University. The new medical school was entirely of Brookings's creation; his was the mind that conceived the development of a school that should supply the Southwest with well-educated graduates in medicine, and his was the driving force that put it through.

When Abraham Flexner, during the course of his investigations of medical schools, inspected the old school at St. Louis, he gave Brookings, who was then president of the corporation of Washington University, the alternative of closing the school or reorganizing it from top to bottom in accordance with modern standards. Brookings saw the justice of Flexner's criticisms and accepted the latter choice. How he did it is told by Hagedorn in his biography.

Brookings was a truly great man, able to see things clearly and to throw himself heart and soul into whatever he undertook. His other achievements, including his services to the nation during the War, are equally inspiring to the reader. His rise from a clerkship in a woodenware house to the position of responsibility that he gladly assumed and gallantly sustained constitutes an epic of American life.

The Diagnosis and Treatment of Pneumonia. Campbell P. Howard. Edited by Henry A. Christian. 373 pp. New York: Oxford University Press.

This volume is a third reprint, the original of which appeared in 1931 as the tenth and last of the series of Oxford monographs edited by Dr. Christian. In the section on Lobar Pneumonia, which occupies two-thirds of the book, there has been inserted a total of 40 pages, and the bibliography has been extended to correspond. The additional pages deal with the recent advances in bacteriologic classification and in treatment.

The material in this book is presented clearly and authoritatively by an eminent clinician and manifests, in many respects, the influence of Osler, under whom the author received his early clinical training. Laboratory aspects are dealt with adequately. Such recent subjects as the use of artificial pneumothorax and the extension of specific treatment to cases due to the newer types of pneumococci have been inserted.

The volume is well bound, clearly printed and, while best suited for the physician engaged in the practice of internal medicine, can be read with profit by all physicians and specialists alike. Some of the recently inserted pages contain charts and tables not adequately described and not entirely in keeping with the tone of the rest of the volume.

Applied Dietetics for Adults and Children in Health and Disease. Sanford Blum. 408 pp. Philadelphia: F. A. Davis Company. $4.75.

The book is essentially a formulary of foods in two parts; one is for adults and the other for children. Healthy states as well as most diseases and disorders are listed alphabetically; and under each heading the foods to eat and to avoid are recorded, the regime for a day is given, and special considerations are described.

Under "normal diet," steaks, roast beef, lamb, oysters and liver are some of the proteins to eat; and tongue, kidneys, liver, sausage, pork and lobsters are some of those to avoid. Corn, cabbage, sprouts and turnips are some of the vegetables to avoid; and nuts, pies, cakes, waffles and sauces are some of the sundry foods not to consume. Under the heading, "type régime for a day," a somewhat limited list of foods to eat for breakfast, lunch and dinner is given. Some special considerations are to eat slowly, only at meals and to omit all gravies, condiments and greasy foods. While the special considerations given for the consumption of food are generally sound, the foods allowed a healthy person seem somewhat restricted. The position of liver is anomalous, since you can and you cannot eat it. This may be a misprint. The book may be found useful for practitioners and dietitians.

Skin Diseases in Children. George M. MacKee and Anthony C. Cipollaro. 345 pp. New York and London: Paul B. Hoeber, Inc. $5.50.

The title "Skin Diseases in Children" describes this book very well. The range of both common and rare diseases in children is covered most adequately and even an occasional disease almost never seen in children is included. With only one other book devoted to skin disease in children, and that a part of a system of pediatrics, this book should fill a definite place in the libraries of general practitioners and pediatricians especially. It is essentially a practical book, reviewing the manifestations of the various diseases and going into details of treatment, often with a brief statement regarding etiology. There are also many excellent illustrations taken from the very large collection of the authors. It might have been well to have placed the chapter concerning the care of the normal skin first and to have devoted somewhat more time to its discussion. Syphilis, as the authors indicate, cannot be adequately covered in a small volume of this sort, but a general outline of the features of this disease does as well as could be accomplished in such a short space. All in all, the book covers the field of children's skin manifestations very efficiently.